A. M. A.
ARCHIVES OF
NEUROLOGY AND PSYCHIATRY

INDEX NUMBER

JUNE 1952
VOLUME 67 NUMBER 6

Published Monthly by
AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter Jan. 7, 1919, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $12.00

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THE A. M. A. ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published by the American Medical Association to stimulate research in the field of diseases and disorders of the nervous system and to disseminate knowledge in this department of medicine.

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The A. M. A. ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published monthly. The annual subscription price (for two volumes) is as follows: domestic, $12.00; Canadian, $12.40; foreign, $13.50, including postage. Single copies are $1.25, postpaid.

Checks, money orders and drafts should be made payable to the American Medical Association.

A. M. A.
Archives of Neurology and Psychiatry

VOLUME 67 JUNE 1952 NUMBER 6

ABSENCE OF CLINICAL EVIDENCE OF DESTRUCTIVE LESIONS OF THE SYMPATHETIC NERVOUS SYSTEM IN ACUTE ANTERIOR POLIOMYELITIS

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THERE is need for clarification of the reports upon disturbances in functions of the sympathetic nervous system in acute anterior poliomyelitis. Often coupled with reference to the work of others, and with reports of authors themselves upon pathologic changes in the intermediolateral column of cells and in the ganglia of the sympathetic chain, is a description of some change in function of the sympathetic nervous system, leading to the assumption that the dysfunction noted is related to the pathologic changes described. For the most part, evidence of such dysfunction has consisted of some one sign or symptom, such as excessive sweating, increased electrical skin resistance in the axillae, or temporary retention of urine during the acute stage. At times more comprehensive studies have been made, including determinations of skin temperature and oscillometry, during the acute stage, as well as late after the onset of the disease.[1]

Far from demonstrating evidence of a destructive lesion in the sympathetic nervous system, most of the reports have indicated an integrity of the sympathetic nervous system. Thus, the hyperhidrosis reported in the early stage clearly indicates its integrity, whereas, at the same time, an increase in skin temperature might be related to its paralysis. Simultaneously, it is reported by many authors that vasospasm exists. This certainly does not indicate a destructive lesion of the

This study was aided by a grant from the National Foundation for Infantile Paralysis.

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Read in part at the Seventy-Sixth Annual Meeting of the American Neurological Association, June 19, 1951, Atlantic City, N. J.

1. Zellweger, von H., and Morf, H.: Helvet. paediat. acta **5**:434, 1950.

sympathetic nervous system, but, rather, suggests an overaction of it. Vasospasm has been described by some as occurring during both the acute and the late stage of the disease, and the treatment advocated has been use of sympathicolytic drugs and sympathectomy.

Some accredit the changes in sympathetic function to a hypothetical imbalance. One proposed explanation is that there exists an irritative locus in the periphery and that when it persists "a reflex arc producing pain and spasm is established. This arc may perpetuate itself through the internuncial pool and eventually involve the sympathetic neuron cells, in the lateral horns." We have been unable to find any evidence for the validity of such a philosophical theory.

We should expect a diminution or loss of function to result from pathologic change in the sympathetic nervous system, and should expect reflex imbalance only if suprasegmental regulatory impulses were interrupted. These situations are analogous to what occurs when the lower motor neurone is diseased and paralysis ensues, and when the corticospinal tract is involved and spasticity is present. In relation to motor function, it has been proposed that lesions of internuncial fibers may result in an upper-motor-neurone defect other than that subserved by the corticospinal tract. As a result, inhibitory impulses may not reach the anterior horn cells, and spasm occurs. Were a similar situation present in respect to the sympathetic nervous system, a reflex overaction of its functions would be expected to result from suitable stimuli, as is the case in transverse lesions of the cervical portion of the spinal cord, when reflex hypertension and excessive sweating result from stimulation by bladder distention, and reflex hypertension from stimuli of cold. Although we have not found any evidence in the literature supporting this theory, it is possible that, for some reason, facilitatory or inhibitory impulses might be interrupted, as suggested by rare observations in two of our cases.

Pathologic changes in the sympathetic nervous system have been found by various investigators. Kuntz [3] stated:

> In cases of poliomyelitis, muscular paralysis is accompanied by segmental vasomotor and sweat secretory disturbances. In this disease, the inflammatory process in the spinal cord may involve the intermedio-lateral cell column directly, but not infrequently pathologic changes also occur in the corresponding ganglia of the sympathetic trunk.

The latter alterations have again been described by Smith and his co-workers.[4] However, Bodian,[5] in reviewing 24 fatal cases, found the intermediolateral column of cells little involved except in one case in which there was considerable destruction. Lesions which could affect internuncial fibers in the spinal cord certainly have been demonstrated, as well as lesions in the reticular formation of the brain stem which might result in changes in function of the intact sympathetic nervous system.

Two major problems presented themselves. Was there any consistent clinical evidence that a possible destructive lesion of the sympathetic nervous system, whether in the intermediolateral column of cells or in the ganglia of the sympathetic

2. Smith, E.; Graubard, D. J.; Goldstein, N. P., and Bikoff, W.: New York J. Med. **48**:2608, 1948.

3. Kuntz, A.: The Autonomic Nervous System, Ed. 3, Philadelphia, Lea & Febiger, 1945.

4. Smith, E.; Rosenblatt, P., and Lemaruro, A. B.: J. Pediat. **34**:1, 1949. Smith, E., and others: J. A. M. A. **144**:213, 1950.

5. Bodian, D.: J. A. M. A. **134**:1148, 1947.

chain, results in paralysis of function? Was there any clinical evidence for the assumption that reflexes subserved by the sympathetic nervous system were either suppressed or increased as the result of suprasegmental lesions?

MATERIAL FOR STUDY

During the epidemic of anterior poliomyelitis in the fall of 1950, Dr. Herman N. Bundesen, president of the Chicago Board of Health, granted us permission to conduct a study upon the patients in the Municipal Contagious Disease Hospital of Chicago. In this study, Dr. Mark H. Lepper, medical superintendent of this hospital, gave us liberal assistance and cooperation.

In the plan of the study, since the intermediolateral cell column extends from the eighth cervical to the second lumbar segment, inclusive, only patients the distribution of whose pronounced paralysis indicated severe involvement of contiguous anterior horn cells were to be used for final analysis. Thus, for the thoracic outflow, paralysis of the upper intercostal muscles and the distal portions of the upper extremities and, for the lumbar outflow, paralysis of the lower intercostal muscles and complete paralysis of muscles of the lower extremities innervated by the upper lumbar segments, were required. Ninety-one patients were surveyed for study during the epidemic in the fall of 1950 at the Municipal Contagious Disease Hospital of Chicago. Some patients died too soon to permit examination; others were too ill and in a respirator; still others recovered, when earlier observation promised the development of a severe paralysis. Because of the method of selection and the aforementioned circumstances, 24 patients were finally selected for analysis.

In 11 patients all extremities were paralyzed; in 4, both lower extremities; in 3, one lower extremity; in 2, one upper extremity; in 2, both lower extremities and one upper extremity; in 1, one upper and one lower extremity, and in 1, two upper extremities, for a total of 67 paralyzed extremities. We defined the signs of destruction of function of the sympathetic nervous system as consisting, not of some differences in temperature alone or of skin resistance alone or of sweating alone, but of absence of function of the sympathetic nervous system in all its modalities in the dermatomes being studied. This criterion was decided on because, for many reasons, in one or another area there may be a difference in some single function. The material was to be analyzed first in respect to skin temperature, sweating, and skin resistance. Oscillometric measurements were to be added, since they have been reported in the studies of others. Moreover, further to determine the integrity of vasoconstriction and possible reflex hyperreaction, cold pressor tests were performed. In a few other cases at the United States Veterans Administration Hospital, Hines, Ill., the effect of bladder distention was studied.

METHODS OF EXAMINATION

Skin temperatures were measured with a U. M. A. Inc. thermocouple at room temperature after exposure of the body for 20 minutes. In some respirator patients this was impossible. This study was made to determine whether any significant difference in rise of skin temperature could be detected.

A profile of a curve obtained for the normal subject with the skin temperature plotted against areas beginning with the forehead and descending to the fingers, and against areas beginning in the trunk and descending to the toes, has been reported to show a gradient with a higher value in the proximal and a lower value in the distal portion of the extremities. After sympathectomy, this gradient disappears, and the skin temperature of all areas is approximately the same. The presence or absence of such a gradient was sought in our cases.

Sweating was observed, and the relative humidity of the skin was measured by the American Instrument Company-Dunmore Electric Hygrometer Indicator. This apparatus, which employs a Hygro-Cel as the sensitive element, was devised to measure the water content of commercial products, such as soap. It was found to be very useful in the study of sweating. Readings were taken every 5 seconds up to 60 seconds after application, and the angle of change, as well as the relative humidity, as expressed on the scale of the galvanometer, was used. This method obviates the messy technique involved in dye studies.

Measurements of electrical skin resistance were made with an R. C. A. ultrasensitive meter, which has megohm ranges. It is a three-stage vacuum-tube voltmeter employing negative feedback for stabilization. The maximum voltage that it could apply to the patient was ½ volt, and this only on the areas of highest resistance. This type of megohmeter was chosen in preference to the bridge type because of the greater sensitivity, more rapid measurement, and lower voltage applied to the patient. Bipolar electrodes at a fixed distance were employed to effect uniform area and spacing. Of course, one cannot measure true electrical resistance by means of any direct current ohmeter, and apparent skin resistance will vary quantitatively with each ohmeter used. Changes in polarization occur which reduce the apparent skin resistance, and to obviate this factor readings were taken 30 seconds after application of the electrodes.

The Collens Sphygmo-Oscillometer was used for the study of the amplitude of oscillations. The data obtained by oscillometry are in general less significant than those obtained by thermometry. The oscillations vary from time to time and from person to person. However, two comparisons may be valuable in respect to discovery of sympathetic denervation. First, a difference exists between normal and denervated extremities wherein, on the denervated side, there is a greater amplitude of oscillations; second, a gradient exists wherein the amplitude of the oscillations in the leg is greater than that in the forearm.

Reflex changes in blood pressure after immersion of an extremity in ice water were studied. When the thoracic outflow was to be studied in paralysis of upper extremities, a foot was immersed; and when the lower extremities were paralyzed, a hand was immersed. When only one extremity was paralyzed, if it was an upper one, the contralateral foot was immersed; and if it was a lower one, the contralateral hand was immersed.

As was the case with skin temperature, so with electrical skin resistance and sweating, the profile of a curve plotting areas against observations will not be found to be a plateau. Variations will be found from one to another part of the body. We have not attempted to compare electrical skin resistance or sweating in one or another area in these cases with similar areas in the normal subject. Therefore, the total number of areas in which high skin resistance was found or sweating was absent will be greater than actually would be the case if this were compared with gradient charts.

RESULTS

General Analysis

Among the 24 patients found suitable for study, 5 showed no deviation from normal of skin resistance or spontaneous or heat-induced sweating, or increase in skin temperature. Three of these patients had paralysis of all extremities and of the chest and were in respirators; one had paralysis of one upper extremity, and one had paralysis of an upper extremity and an ipsilateral lower extremity.

In one patient the only abnormality was an increase in skin temperature of the face. This patient had paralysis of all the extremities and the chest.

In four patients the only deviation was dryness of the skin. This occurred symmetrically in extremities whether they were paralyzed or unparalyzed. Two had paralysis of both lower extremities, and two, paralysis of only one lower extremity. In all four patients heat produced sweating in all extremities.

In six patients there was a high skin resistance coupled with dryness of the skin. Both changes were observed in paralyzed and in unparalyzed extremities.

In one patient with paralysis of all extremities, the skin of the face, although dry, had a low electrical resistance, and the skin of both forearms was moist, although its electrical resistance was high.

In another patient, with paralysis of the right lower extremity alone, skin resistance was high and the skin dry in the upper, unparalyzed extremities, whereas in the paralyzed lower extremity skin resistance was low, despite dryness of the skin. In another patient, with paralysis of the upper extremities and chest, in the

paralyzed extremities the skin resistance was high, and the skin was dry except in the face and palms; in the legs, although the electrical resistance was high the skin was moist.

In another patient, with paralysis of all extremities, the skin resistance was high and the skin dry in all extremities, but the face, although dry, had a low skin resistance.

In another patient, with paralysis of the lower extremities, dryness of the skin was coupled with high skin resistance in both paralyzed and unparalyzed extremities, except for the palms, soles, and deltoid areas, where, although the skin was dry, the skin resistance was low.

The remaining three patients had paralysis of all the extremities. In the upper ones, dryness of skin was coupled with high skin resistance except in the palms, and the same was true of the face; in the lower extremities the skin resistance was low, despite a dry skin.

Heat Sweating.—In all patients sweating occurred after heating, and there was no difference between the paralyzed and the unparalyzed extremities. Three of this group with paralysis of the upper extremities and chest were treated in a respirator. In eight patients, there was failure on the part of 1 or more areas to sweat after heating; in three patients, in 2 of 20 areas examined; in two, in 5 areas; in one, in several areas; in one in 12 areas, and in one, in 14 areas.

In the first patient, with paralysis of both lower extremities, only the soles failed to sweat after heat. The skin resistance was low throughout except on the soles, although the skin was dry. There was a good temperature gradient in the extremities, and the oscillometric ratio was normal.

In the second patient, with paralysis of both lower and the right upper extremity, the dorsum and sole of the right foot failed to sweat, the skin resistance being low in the paralyzed lower extremities and high in the paralyzed and unparalyzed upper extremities. There was a good temperature gradient.

In the third patient, with paralysis of all extremities, the anterior part of the right leg and the right sole did not sweat after heating. A temperature gradient was present.

In the fourth patient, with paralysis of both lower extremities, the anterior surface of the left leg and the dorsum and sole of both feet failed to sweat. Skin resistance was low throughout. There were a temperature gradient in both paralyzed extremities, a rise in blood pressure in the cold pressor test, and an oscillometric ratio of 1.4 on the right and of 2 on the left.

In the fifth patient, with paralysis of all extremities, there were five areas in which heat sweating was absent—the right forearm, the left arm and forearm, and both soles. The skin resistance was low in the soles and, for the most part, in the lower extremities. There were a temperature gradient in the lower extremities and a normal oscillometric ratio.

In the sixth patient there was paralysis of only the left upper extremity. On the right, unparalyzed side, in the area of the arm, tibial region, and sole, no sweating occurred as the result of heat. On the left side there was no sweating on the sole, tibial area, arm, and shoulder. Of seven areas with absence of heat sweating, two occurred in the paralyzed extremity and five in unparalyzed areas; in three the

skin resistance was high. There was no difference between the unparalyzed (right) and the paralyzed (left) upper extremity with respect to skin resistance, skin temperature, or oscillometric ratio.

The next two patients had paralysis of both lower and upper extremities. They showed the largest number of areas in which heat sweating was absent, 12 and 14 areas, respectively. In one patient all the eight areas in the upper extremities failed to sweat, the absence in sweating being associated in three with high skin resistance. However, both sides of the face had heat sweating and low skin resistance. In the lower extremities, four areas failed to sweat, and in one the skin resistance was high. Oscillometric readings were normal, and a gradient of skin temperature was recorded in the lower extremities. In the last patient, with the exception of the face, over which sweating occurred and skin resistance was low, all areas of the upper part of the body failed to sweat, and all areas on the lower extremities as well. There was no rise in skin temperature, and a good temperature gradient was observed in the lower extremities.

Summary.—Five patients were without any change. Eleven patients showed the following changes: One had only an increase in skin temperature of the face. Four had dryness of the skin only, which occurred in paralyzed, as well as in unparalyzed, extremities. In six there was high skin resistance, coupled generally with dryness of the skin; there was no difference between paralyzed and unparalyzed extremities, and in all patients heat sweating occurred in the theretofore dry areas of skin.

The last eight, in whom heat sweating was absent in one or more areas, deserve further comment. In the other patients the dorsum and sole of the foot often were dry in paralyzed, as well as in unparalyzed, extremities; and in these 8 patients absence of sweating was most frequently encountered in these areas, the sole being affected in 12 of 14 extremities and the dorsum of the foot in 7 of 14 extremities. The small number of areas involved in all but two patients of this group and the fact that in all skin temperatures, reactions to the cold-pressor tests, oscillometric readings, and low skin resistance failed to reveal differences between paralyzed and unparalyzed limbs, and, at times, showed a preponderance of failure to sweat on the part of the skin on the unparalyzed side, serve to indicate that the failure of heat sweating was in these cases not due to sympathetic denervation.

Only the last two cases, at first glance, seem more convincing. In both there was paralysis of all extremities. In the first, except for the feet, the areas with absence of heat sweating were limited to the upper extremities, but of eight such areas, only two had a high skin resistance; the face showed heat sweating, and the skin resistance was low; moreover, a temperature gradient was present in the lower extremities. There was no significant rise of temperature. In the last case, except for the face, where heat sweating occurred, all areas of the body remained dry. However, there was no significant rise in skin temperature, and a good skin-temperature gradient was observed in the lower extremities.

Although in the last two cases, especially in the last, absence of heat sweating was suggestive of denervation, the integrity of the sympathetic nervous system was established by other tests. However, both the patients, in addition to being paralyzed in all four extremities and the chest, had bulbar involvement, as indicated by hoarseness and dysphagia in one and by relatively early central paralysis of respiration in the other. This is suggestive that central heat-regulatory impulses from the hypothalamus may have been interrupted.

Statistical Analysis

The impression gained from the clinical review may be confirmed by a statistical study.

In the 24 patients for whom the data are analyzed, 576 areas were each examined for increase of skin temperature, high electrical skin resistance, and absence of moisture. These areas included the forehead, face, shoulder, arm, forearm, palm, chest, abdomen, thigh, leg, and dorsum and planta of the foot on the right and left sides. A total of 2,304 observations were made on 576 areas.

Of 1,480 observations on paralyzed extremities, 301 (20.33%) indicated a possible change in function of the sympathetic nervous system, and of 824 observations on unparalyzed extremities, 173 (20.99%) gave such an indication. The incidence of variation from the expected normal was the same in paralyzed and in unparalyzed extremities.

If any segments of skin were deprived of sympathetic nerves, it would be expected that in these areas all the methods of examination of function of the sympathetic nervous system would yield findings consistent one with the other, and

Percentage of Areas in Paralyzed and Unparalyzed Extremities Showing Loss of Varying Numbers of Sympathetic Functions

No. of Functions Damaged	Percentage of Areas Examined	
	Paralyzed Extremities	Unparalyzed Extremities
None	42.2	32.5
One	36.2	52.4
Two	19.0	13.6
Three	1.9	1.4
Four	0	0

occurring concurrently. If the skin resistance were high, there should be absence of spontaneous or heat-induced sweating, accompanied by a significant rise in temperature and absence of a skin-temperature gradient.

To determine the consistency of the findings, we first estimated the numbers of areas in which only one of the four methods of examination yielded information suggesting paralysis of function of the sympathetic nervous system, and how many revealed a combination of two, three, or four positive results (Table). One function alone was affected in 36.2% of areas examined in paralyzed extremities and in 52.4% areas in unparalyzed extremities. Two functions were affected together in 19% of areas in paralyzed extremities and in 13.6% of areas in unparalyzed extremities. Three functions were affected simultaneously in 1.9% of areas in paralyzed extremities and in 1.45% of areas in unparalyzed extremities. In none were all four functions affected.

In 42.17% of areas in paralyzed and in 32.53% of areas in unparalyzed extremities there was no evidence of dysfunction.

Dryness of the skin alone occurred in 22.2% of areas in paralyzed and in 45.6% of areas in unparalyzed extremities.

Skin resistance was high and constituted the only sign in 10.8% of areas studied in paralyzed extremities and in 5.8% of areas in unparalyzed extremities.

Heat sweating alone was absent in 2.2% of areas in paralyzed and in 1.0% of areas in unparalyzed extremities.

Increase of skin temperature alone was present in 1.1% of areas in paralyzed and in none of the areas in unparalyzed extremities.

Dryness of skin occurred more frequently as a single sign in unparalyzed extremities, and increased electrical skin resistance, more frequently in paralyzed extremities. Heat sweating was absent in very few areas, and increased skin temperature only was present in the face of one patient.

When we estimated the percentage of areas and of observations on any deviation from expected normal, whether occurring singly or together, we found that a high electrical skin resistance was present in 32.9% of the 301 areas on paralyzed extremities and in 22.5% of the 173 areas on the unparalyzed extremities.

Absence of spontaneous sweating was observed in 52.1% of the areas on paralyzed extremities and in 72.2% of the areas on unparalyzed extremities.

Heat-induced sweating was absent in 13.6% of areas on the paralyzed and in 5.2% of areas on the unparalyzed extremities.

Elevated skin temperature was found in 1.3% of areas on the paralyzed and in none of the areas on the unparalyzed extremities.

As to heat sweating, it was found that when the response was evaluated with respect to possible denervation, extremities instead of scattered areas being used for analysis, absence of heat sweating in two patients was not supported by other evidence of denervation; but the generalized absence of heat sweating in one might suggest interruption of heat-regulatory impulses from the hypothalamus.

Summary.—It may be said that a single indication of possible dysfunction occurred in significant numbers and was more frequently found in unparalyzed extremities. A combination of two indications was found in too few cases, and with only slightly greater frequency in paralyzed extremities, to be significant. In a very few cases a combination of three indications was found about equally in paralyzed and in unparalyzed extremities. In no case was there found a combination of all four indications of possible dysfunction of the sympathetic nervous system.

Comparison of the paralyzed upper extremity with the opposite, unparalyzed one showed no significant difference in areas involved or in numbers of deviations from normal. The same was true when one compared a paralyzed lower extremity with an opposite, unparalyzed one. Comparison of a paralyzed upper extremity with an unparalyzed lower extremity showed a slightly greater number of areas involved and a greater number of areas with deviations from normal on the paralyzed upper extremity. Comparison of a paralyzed lower extremity with an unparalyzed upper extremity showed a larger number of areas involved and of deviations from normal on the unparalyzed upper extremity. Comparison of paralyzed with unparalyzed extremities showed that 72.1% of 122 areas examined in paralyzed extremities and 68.6% of 140 areas in unparalyzed extremities gave evidence of some deviation from normal function.

Of 488 potential deviations from normal on the paralyzed extremities, such a deviation was shown in 23.6%. Of 560 potential deviations on the unparalyzed extremities, 21.3% showed such a deviation.

SKIN-TEMPERATURE GRADIENT

Although a gradient in terms of degrees (Fahrenheit) was found to be present in all the lower extremities examined, we were unable to confirm the regular occurrence of a gradient in the upper extremities. Of nine patients in whom the upper extremities were not paralyzed, there was a significant gradient of 8.5 degrees in only one, no gradient in six, and a gradient of 1.5 degrees in one; in one patient the temperature of the face was 8 degrees greater, for no explicable reason.

Of patients with complete paralysis, four upper extremities had gradients of 13, 10, 7, and 4 degrees, respectively, with an average of 8.5 degrees; in 10 patients with complete paralysis of the lower extremities, there were gradients of 18, 11.5, 9, 5.5, 5, 4.5, 3, 2, and 1.5 degrees, respectively, with an average of 6.5 degrees. Of eight patients with incompletely paralyzed lower extremities there were gradients of 14.5, 13, 12, 11, 7, 4, 3, and 2.5 degrees, respectively, with an average of 8.3 degrees.

Another study consisted of comparing the skin temperature of one paralyzed extremity with that of an opposite, unparalyzed one. Of six such comparisons of upper extremities, three showed no difference. In one, there was a greater temperature of the face, arm, forearm, and palm on the unparalyzed side. In another, there was an increase of temperature of the arm, forearm, and palm, again, of the unparalyzed side. Only in one was there a higher temperature in the paralyzed extremity, and that only in one area, the forearm.

In four patients in whom comparisons of the lower extremities could be made, there was no difference in skin temperature between paralyzed and unparalyzed extremities.

OSCILLOMETRY

Of 68 extremities, oscillometric pulsations were studied in four areas: the arm, forearm, thigh, and leg.

Of 10 paralyzed extremities which could be compared with an opposite unparalyzed extremity, there was no difference in amplitude of oscillometric pulsations in 4; in 3 the amplitude was greater on the paralyzed side, and in 3, on the unparalyzed side.

The ratio of the amplitude of oscillations in the leg to that in the forearm is said to be from 1:2. If the oscillations were of greater amplitude when the lower extremities only were denervated, this index would be greater. Of 11 such extremities examined, in only 1 was the ratio increased, with an index of 3. No other findings supported the conclusion of denervation. In these 11 extremities, the indices were 1.0, 1.0, 1.0, 1.0, 1.4, 1.6, 1.6, 1.6, 1.66, 2, and 3, respectively.

COLD-PRESSOR TEST

Of 15 patients with paralysis of the upper extremities, there was a fall of systolic blood pressure in 2; in 1 there was no response in an opposite, normal extremity. In the others a rise in blood pressure occurred. The range was from -10 to $+30$ mm., with a median of $+10$ mm. The response in diastolic pressure was a rise in pressure in seven, no response in four, and a drop in three, with a median of 0 and a range of -6 to $+30$ mm. There was no consistent difference between paralyzed and unparalyzed extremities.

Of 11 patients with paralysis of the lower extremities in whom a study could be made, the systolic blood pressure dropped in 2, and in 1 there was no response; the range was from — 10 to + 20 mm., with a median of 10 mm. The diastolic pressure dropped in one; in five there was no response. Of three in whom the diastolic pressure had not risen, there was an increase of systolic blood pressure in one, a fall in one, and no change in the third. There was no consistent difference between the paralyzed and the unparalyzed side. The range of change of systolic pressure was from — 5 to + 20 mm., with a median of 0.

In normal man, the range is from 0 to 20 mm., and in hyperreactors, above 20 mm. In traumatic lesions of the cervical spinal cord in which inhibitory impulses fail to reach the sympathetic outflow, there is an excessive rise of blood pressure upon stimulation of an extremity with ice water—increases as high as 40 to 60 mm. Hg having been observed. In this connection, in another study on patients in the residual state of poliomyelitis a few months after onset of the disease, we found two in whom the cold-pressor test resulted in a rise of systolic blood pressure of + 52 and + 47 mm. Hg, respectively. In one it could be interpreted as evidence that the patient was a hyperreactor with initial hypertension. In the other the rise suggests that inhibitory fibers in the reticular formation of the brain stem or elsewhere may be affected and reflex hypertension result. However, the case is further proof that there is no destructive lesion in the sympathetic nervous system itself. This single instance of reflex hypertension also indicates the rare occurrence of the phenomenon.

We have not observed reflex hypertension from bladder distention in four cases in which it was sought.

SUMMARY

Ninety-one patients suffering from acute anterior poliomyelitis during the fall of 1950 were examined at the Chicago Municipal Contagious Disease Hospital. Of these, 24 patients met the requirements necessary for this study.

In 11, all extremities were paralyzed; in 4, both lower extremities; in 3, one lower extremity; in 2, one upper extremity; in 2, both lower and one upper extremity; in 1, one upper and one lower extremity, and in 1, both upper extremities. In all, there were 67 paralyzed extremities.

The studies conducted on these patients consisted of measurement of skin temperature, electrical skin resistance, and sweating; determination of the relative humidity of the skin by a hygrometer; oscillometry, and cold-pressor tests. In the study of skin temperatures, a significant increase in skin temperature, differences between paralyzed and unparalyzed extremities, and a skin-temperature gradient were sought. In the study of amplitude of vascular oscillations, the ratio of the amplitude of the oscillations in the leg and that of oscillations in the forearm was estimated; similarly, the ratio for paralyzed and unparalyzed extremities was determined. In respect to electrical skin resistance and sweating, the measurements on the paralyzed side were compared with those on the unparalyzed side. Finally, when any one of these methods served to indicate a dysfunction, effort was made to determine whether such a conclusion could be confirmed by the other methods of examination.

In five patients there was no indication of any change from the normal. In 11 patients such changes as consisted of dryness of the skin alone or dryness

coupled with high skin resistance in areas were scattered about equally over paralyzed and unparalyzed extremities; there was no difference in skin temperature, and all the dry areas of skin exhibited heat sweating. In six patients heat sweating was absent in a few areas, at times predominantly on the unparalyzed side. The concept that there was sympathetic denervation was not supported by changes in skin temperature, cold-pressor tests, oscillometry, or often by the presence of high electrical skin resistance.

It is well known that changes in vasomotor, pilomotor, and sweating functions are observed in acute anterior poliomyelitis. However, they are also observed in many infectious diseases of the central nervous system at times when the pathologic changes are limited to the meninges, and at times to the cerebrum alone. At a later stage, such changes as have been reported are those of vasoconstriction and lowering of skin temperature, and do not differ from those observed in extremities the mobility of which is diminished, for example in arthritic limbs particularly those with periarticular atrophies. Such isolated changes as we have described above we believe are related, not to denervation of the sympathetic nervous system, but to reflex changes.

The last two cases deserve additional attention; in them the failure of heat sweating was more generalized. In neither case did the tests support a conclusion that there was sympathetic denervation. Since both were complicated by bulbar involvement, one demonstrating dysarthria and dysphagia, and the other an acute central respiratory paralysis, there is a possibility that heat-regulatory impulses from above were interrupted by lesions in the brain stem from reaching the sympathetic outflow.

In summary, it may be said that a single indication of possible dysfunction occurred in significant numbers and was found more frequently in unparalyzed extremities, that a combination of two indications was found in too few numbers, and with only slightly greater frequency in paralyzed extremities, to be significant, that a combination of three indications was found with about equal frequency in paralyzed and in unparalyzed extremities in very few instances, and that in none was there a combination of all four indicators of possible dysfunction of the sympathetic nervous system.

The impression gained from this clinical review may be confirmed by a statistical study.

In each of the 24 cases analyzed, a total of 576 areas were examined for skin temperature, electrical skin resistance, and moisture. These areas included the forehead, face, shoulder, arm, forearm, palm, chest, abdomen, thigh, leg, dorsum, and planta of feet on the right and left sides. A total of 2,304 observations were made on the 576 areas.

Of 1,480 observations on paralyzed extremities, there was an indication of some possible change in function of the sympathetic nervous system in 301, or 20.3%, and of 824 observations on unparalyzed extremities such an indication occurred in 173, or 21%. The incidence of some change from the expected normal was the same in paralyzed and in unparalyzed extremities.

In a comparison of the paralyzed and the unparalyzed extremities, 72.1% of 122 areas examined in paralyzed extremities and 68.6% of 140 areas in unparalyzed extremities gave evidence of some deviation from normal function. Of 488

potential deviations from normal on the paralyzed extremities, 23.6% showed such a deviation, and of 560 potential deviations from normal on the unparalyzed extremities 21.3% showed such a deviation.

There was no significant or consistent difference in skin temperature between paralyzed and unparalyzed extremities.

In 68 extremities, oscillometric pulsations were studied on four areas—arm, forearm, thigh, and leg. There was no significant difference in amplitude between paralyzed and unparalyzed extremities. The oscillometric ratio was normal in all but one case, and no other evidence of denervation was found in this case.

In the cold-pressor test there was no consistent difference in the response of increase in blood pressure between paralyzed and unparalyzed extremities. Of 15 patients in whom the upper extremities were paralyzed, there was a rise in blood pressure in 13. Of 11 patients with paralysis of the lower extremities, there was a rise in 8, quite within normal limits.

In two patients examined elsewhere, several months after the acute attack, the systolic blood pressures rose 47 and 52 mm. Hg, respectively. The patient who had hypertensive disease was probably a hyperreactor; the reading for the other suggests the possibility of interruption of inhibitory impulses from above. In four patients tested for changes in blood pressure after distention of the urinary bladder no change was found.

Despite the reports of pathologic changes in the sympathetic ganglia, our study of patients suffering from acute anterior poliomyelitis by means of hygrometry, oscillometry, skin-temperature readings, measurements of electrical skin resistance, observation of sweating, and cold-pressor tests failed to reveal any clinical evidence of destruction of function of the sympathetic nervous system.

RESTORATION OF FUNCTION THROUGH NEUROMUSCULAR REEDUCATION IN TRAUMATIC PARAPLEGIA

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IN SERIOUS accidents resulting in fracture or dislocation of one or more vertebrae, the spinal cord may also be injured. In complete transection of the spinal cord, all voluntary motion and sensation are lost permanently below the level of the injury. In partial transection of the spinal cord, function below the injury may be absent at first and return to a variable extent, depending on the severity of the damage to specific tracts. The spontaneous return of function may occur in the course of a few weeks but sometimes takes a number of months. It is generally agreed that if no voluntary motion or sensation has returned in the course of a year after the injury there is no expectation that any function can ever be restored.

If, in the original injury, the spinal cord was completely severed, there is no hope at present of restoration of voluntary motion or sensation in the segments below the transection of the spinal cord, because the evidence is conclusive that regeneration of such a transected spinal cord does not occur in man. If, in the original injury, the spinal cord was not severed completely but early laminectomy was not performed, the compression of the spinal cord by hemorrhage and other factors could conceivably destroy the remaining intact cord tissue at this point by ischemia over a relatively short period. The possibility of salvaging some remaining cord tissue through decompression and improvement in circulation has led, correctly, to strong advocacy of early laminectomy in such cases.

While the differentiation of complete from partial transection of the spinal cord would appear to be relatively simple, considerable difficulty is encountered clinically in this procedure in some cases. The patient with a partial transection of the spinal cord can be observed to perform voluntary motions through contraction of muscles innervated by segments of the cord below the injury and may have conscious localized sensation in response to various modalities of sensory stimuli in root areas below the injury to the cord. In the patient with complete transection, voluntary motion and sensation are entirely absent in segments below the injury to the spinal cord. It is apparent that sufficient time must elapse after the injury before one can be certain that voluntary motion and sensation will not return spontaneously. Furthermore, one must distinguish carefully between true voluntary motion and reflex spasms; the latter may either be elicited or occur spontaneously. Reflex spasms will appear only after recession of spinal shock and must not be mistaken for returning voluntary motion, since spasms can occur through function of the isolated spinal cord completely severed from its brain connections. The earlier belief that the presence of extensor spasm indicated partial transection of the spinal

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cord has been effectively disproved.[1] On the other hand, reflex spasms and mass reflexes can occur in patients with partial transection, as well as in persons with complete transection of the spinal cord. It is just as important not to be confused by involuntary spasms and to find the voluntary motion in patients with partial transection of the spinal cord, so as to be able to give a more hopeful prognosis, as it is to avoid the mistake of considering the reflex spasms an indication of returning voluntary motion and thereby give false hope to a patient with a completely transected spinal cord.[2]

One encounters considerably greater difficulty in clinical testing to be certain that a patient who has no apparent voluntary motion or sensation in segments below the spinal cord injury really has complete severance of the spinal cord. One method to determine this is to inspect the site of injury during the laminectomy. Information gained from this procedure is far from accurate, however, since in many instances it is not possible to be certain whether all the fibers have been severed. At operation, electrical stimulation can be applied to the spinal cord above the apparent transection to determine whether any muscular contraction occurs in segments below the injury.[2]

My colleagues and I have had the opportunity to observe and work with several hundred patients with injuries to the spinal cord resulting from trauma to the spine, mostly from cave-ins and similar accidents. These patients were sent to the Kabat-Kaiser Institute by the United Mine Workers of America Welfare and Retirement Fund. In many of these patients, a number of years after the original trauma, we have found, using the usual methods of testing, no motor or sensory function below the level of the injury to the spinal cord, an indication of complete transection of the spinal cord. On the other hand, when new methods which we have developed for intense excitation of motor centers and pathways in the central nervous system were applied to these patients, slight voluntary motor function below the level of the transection was demonstrated. These methods, which have been described in detail elsewhere,[3] include the use of summation of mass-movement patterns, proprioceptive stimulation, resistance, and rapid alternating antagonist contractions. With these techniques for strong facilitation and overflow in primitive motor patterns, residual dormant motor connections at the level of the injury were stimulated, and the anterior horn cells in the lower segments of the spinal cord discharged impulses which caused voluntary contraction of the muscles. Since only with these new methods of neuromuscular examination was it possible to stimulate these dormant motor mechanisms through the site of injury with sufficient intensity to produce a motor response, this function had remained entirely dormant and was not demonstrable with less effective methods of excitation of voluntary motion. In

1. Macht, M. B., and Kuhn, R. A.: Occurrence of Extensor Spasm in Patients with Complete Transection of Spinal Cord, New England J. Med. **238**:311-314, 1948.

2. Munro, D.: Two-Year End Results in the Total Rehabilitation of Veterans with Spinal Cord and Cauda-Equina Injuries, New England J. Med. **242**:1-16, 1950.

3. Kabat, H.: (*a*) Studies on Neuromuscular Dysfunction: XI. New Principles of Neuromuscular Reeducation, Permanente Found. M. Bull. **5**:3 (Nov.) 1947; (*b*) XII. Rhythmic Stabilization, a New and More Effective Technique for Treatment of Paralysis Through a Cerebellar Mechanism, ibid. **8**:1 (Jan.) 1950; (*c*) XIII. New Concepts and Techniques of Neuromuscular Reeducation for Paralysis, ibid. **8**:3 (July) 1950; (*d*) Central Mechanisms for Recovery of Neuromuscular Function, Science **112**:23-24 (July) 1950.

other words, some motor fibers were anatomically intact in these patients but required such strong stimulation for the production of a response that they had not functioned at all for many years and there was no expectation that spontaneous recovery of function would ever supervene. Intensive daily treatment over many months with the new methods of neuromuscular reeducation for maximal facilitation of motor mechanisms in the central nervous system [3] resulted in steady improvement and striking increase in voluntary motor function below the transection. Restoration of dormant motor function below the transection was of great practical value in improving ambulation and other essential activities.

It should be pointed out that while restoration of function below the level of the injury to the spinal cord was dependent upon the application of techniques of maximal neuromuscular facilitation through proprioceptive mechanisms, a complete and integrated rehabilitation program was essential for practical achievement. This included hospitalization, good nursing care, good medical care and nutrition, and numerous surgical procedures for complications. The program also included training in self-care and gait, gymnasium and mat work, occupational therapy, and vocational guidance and training, as well as worth-while recreation. Perhaps the most important ingredient in the total program is the psychotherapeutic effect of intensive activity under encouragement for the achievement of practical accomplishments which had not been considered possible and the psychotherapeutic effect of the group activities in the rehabilitation center.

After severe trauma to the spinal cord, even when the transection is incomplete, the anatomically intact nerve fibers are not necessarily restored to function spontaneously. During the period of spinal shock the ability of higher motor mechanisms to stimulate the anterior horn cells in segments below the injury is completely in abeyance. After recovery from spinal shock, there are often prolonged disuse enforced by healing of fractures and pain, and inability to sit up and ambulate, as well as weakness from serious infections, decubitus ulcers, anemia, and malnutrition. Synaptic resistance increases greatly during these periods of spinal shock and relatively prolonged, complete disuse, and it is not surprising that the slight residual motor connection from the brain to the lower segments of the spinal cord would remain dormant indefinitely unless treated intensively with methods of strong excitation.

Overflow of nervous excitation from motor mechanisms above the transection is essential in initial stimulation of the dormant motor connection in the injured spinal cord. For example, the patient shows no voluntary muscular contraction in segments below the injury to the spinal cord even when facilitation techniques are used if the motion is confined to the segments below the injury; thus, attempts at voluntary motion of the lower-trunk or hip muscles directly result in failure. On the other hand, the same muscle groups show slight voluntary contraction when maximal voluntary contraction of the neck or shoulder muscles is combined with attempted motion of the lower trunk or hip in mass-movement patterns. Such overflow from motor patterns above the spinal-cord injury is essential in initiating responses in the dormant muscles. After prolonged treatment, the voluntary contraction occurs in the isolated motions below the transection even without facilitation techniques, and this voluntary motor function can be utilized in practical activities, such as walking.

An unexpected finding has been the recovery of sensory function in the course of intensive neuromuscular reeducation in patients with paraplegia. In the cases to be reported, obvious and significant sensory return has occurred in segments below the injury in the course of, and apparently as a direct result of, the neuromuscular-therapy program. In one of these cases the sensory level had remained unchanged for at least six years after the original trauma. The return of sensation is not clearly explained, but may be due to strong excitation of sensory pathways in the course of voluntary motor training, and possibly increased blood supply in the injured area of the spinal cord through nervous activity. Restoration of sensation has been of practical importance, particularly in relation to bladder and bowel function.

The demonstration of dormant motor function below the level of the injury to the spinal cord in patients who have had apparently complete paraplegia for a considerable period has been a relatively common finding in our series of cases. The three cases presented in detail in this paper illustrate our experience, but we have now observed this phenomenon in a much larger series of cases of damage to the spinal cord by compression fracture from mine accidents, automobile accidents, and bullet wounds and from transverse myelitis and spontaneous hemorrhage.

REPORT OF CASES

Case 1.—A man aged 35 was injured on Nov. 20, 1940, when he was caught between moving coal cars in a coal mine in Colorado. He was immediately paralyzed and lost all sensation below the sternum, and the left arm was so severely damaged that it had to be amputated. He also lost all control and feeling in the bladder and bowels. He was placed in a Bradford frame and a body cast for six weeks. Laminectomy was not performed. He has had recurrent cystitis. The patient has been hospitalized ever since the injury. He was admitted to the Kabat-Kaiser Institute on Dec. 24, 1948.

The essential findings on admission were (1) an old fracture-dislocation of the 12th thoracic and 1st lumbar vertebrae with pronounced left lateral subluxation of the first lumbar vertebra on the 12th thoracic vertebra; (2) complete loss of voluntary motion and sensation below the 6th thoracic segment bilaterally; (3) complete loss of sensation and of voluntary control of urination and defecation; (4) amputation of the left arm, leaving a stump of only the head of the humerus, and (5) bladder stone, 4 by 3 cm.

There were no decubitus ulcers or pain. The general health was good. The patient was able to be up in a wheel chair.

In evaluating this patient's prospects for rehabilitation, it was thought that ambulation would be impossible. A tripod swing-to or swing-through gait on crutches was out of the question because of the very high amputation of the left arm. On the other hand, a four-point gait on crutches and braces would require fairly good motion of the pelvis; since this was absent, and the spinal cord had apparently been completely transected for over eight years, it was believed that the patient could not achieve any method of walking. A plan for his rehabilitation, therefore, consisted of (1) treatment of the urinary tract for stone and recurrent infections; (2) fitting of a prosthesis for the left arm and training in its use as a helping hand, with strengthening of the muscles of the left shoulder; (3) construction of a special wheel chair with drives for both wheels on the right side, so that the patient could wheel and steer it himself with the right hand alone, and (4) vocational rehabilitation.

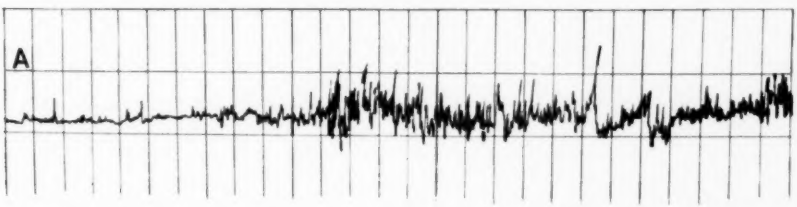
Cystolithotomy and bilateral vasectomy were performed without anesthesia on Dec. 29, 1948.

On April 21, 1949, the patient was checked very carefully again, techniques of maximal facilitation being used in an attempt to obtain overflow for voluntary contraction of lower-trunk muscles below the level of the spinal-cord injury. Using mass-movement patterns of the upper trunk with maximal resistance to voluntary motion of the neck and right arm, very slight flickers of voluntary contraction were palpated in the abdominal muscles and the quadratus lumborum.

The patient was then started on intensive individual physical therapy, two hours a day, for development of voluntary trunk motions below the sensory level, mass-movement patterns of the upper trunk being used for reinforcement. He carried out the neuromuscular reeducation against maximal resistance, using rhythmic stabilization[3b] for further facilitation. Stretch reflexes were also utilized effectively for facilitation, despite the absence of conscious proprioceptive sensation for these muscles, a fact which suggests proprioceptive facilitation at the spinal level.[4] Long aluminum leg braces were also obtained, and he was started on standing in braces in parallel bars, with the therapist helping him, on May 11. He also carried out mat work for sitting balance.

With this program, the patient showed relatively rapid improvement. By Dec. 14, he was walking independently with long leg braces and crutches with the left-arm prosthesis. He was able to elevate the pelvis and clear the floor at each step, using the quadratus lumborum and abdominal muscles, under good voluntary control.

In July, 1950, a new type of prosthesis was designed and obtained for ambulation. This was a massive leather prosthesis which fitted over the left scapula and the upper part of the chest. Instead of the prosthetic arm and hook, a telescoping steel rod fitted into this bucket to serve as a crutch. The patient moved the crutch forward by rotation of the trunk, since the bucket did not allow effective motion of the small stump of the humerus. A large area of the chest was enclosed with a molded bucket, which terminated in 5 in. (12.7 cm.) of artificial upper arm built of balsa wood, reinforced with aluminum, the whole cushioned in sponge rubber. The bucket was supported by a broad leather chest band, approximately 10 in. (25.4 cm.) wide,



Electromyographic records from surface electrodes (Case 1): Action potentials in erector spinae muscle at the second lumbar segment during voluntary extension of the trunk against resistance in a mass-movement pattern.

The patient, a traumatic paraplegic, had had no voluntary motion or sensation below the sixth thoracic segment for 8½ years. The dormant connection for voluntary motion was developed through intensive neuromuscular reeducation. The sensory level remains unchanged, at the sixth thoracic segment.

which encircled the chest and buckled in front with three short straps. Chest expansion was used to draw the device toward the chest wall, so that it was permitted to bear the patient's weight on the crutch. An adjustable ball socket was placed at the bottom of the molded artificial stump. A 1 in. (2.5 cm.) well was bored through the bottom of the ball so that the crutch head could be inserted directly into the ball socket. A snap-ring device permitted rapid insertion or ejection of the crutch. The ball socket is adjustable by means of a series of screws and permits proper alignment by the patient. The crutch itself is of tubular construction, with threaded couplings at the various joints. It collapses to seat level to permit his lifting his weight when sitting, and to floor level when he is standing. The crutch support and the prosthetic arm and hook mentioned above are interchangeable.

The patient continued to improve under the intensive regimen of treatment. He can now (June, 1951) walk independently with long aluminum leg braces, the right crutch and the left crutch-prosthesis and can be on his feet independently for 2½ hours continuously. He has a good four-point gait and clears easily at each step. He can walk slowly approximately four blocks without stopping. He can walk up and down ramps and on grass and rough ground, but cannot go up curbs or steps. He requires only slight assistance under the prosthesis for balance

4. Gellhorn, E.: Proprioception and Motor Cortex, Brain **72**:35 (May) 1949.

in order to rise by himself out of the chair and get on his feet to start walking. Sitting balance has shown striking improvement because of the development of the lower-trunk muscles. He now has fairly good power and endurance in voluntary contraction of the abdominal muscles, quadratus lumborum, lower thoracic muscles, and lumbar sacrospinalis (erectores spinae), which had not functioned at all for the 8½ years following the original injury. There has been no change in the sensory level, with good sensation in the sixth thoracic dermatome and none below this level. He has no feeling of voluntary-muscle contractions of the lower-trunk muscles. There is no feeling or control of the bladder or bowel. There has been no demonstrable voluntary contraction of any muscles in the lower extremities.

In February, 1951, electromyographic records were made of abdominal muscles at the 11th thoracic segment and of the erector spinae muscles at the second lumbar segment, and action potentials were recorded in voluntary contraction of these muscles with facilitation (Figure).

Case 2.—A man aged 36 was run over by a loading machine while working in a mine in Kentucky, on April 22, 1943. He immediately lost all voluntary motion and sensation from the waist down and had incontinence of the bladder and bowel. Within six hours after the injury he had an operation for reduction of the fracture-dislocation of the vertebrae and a laminectomy. There was essentially no return of function from the time of injury to the time of his admission to the Kabat-Kaiser Institute, in California, on Nov. 1, 1948.

The essential findings on admission were (1) x-ray evidence of partial fusion of the 11th and 12th thoracic vertebrae after compression fracture and laminectomy; (2) complete flaccid paraplegia of both lower extremities with no demonstrable voluntary pelvic motion; (3) normal sensation through the 12th thoracic dermatome and no sensation below this level except for a very slight feeling of bladder fulness; (4) incontinence of bladder and bowel with no voluntary control and no feeling of bowel fulness (he had just sufficient feeling of bladder fulness that he would reach for a urinal immediately and not wet himself); (5) talipes equinus of both feet, due to shortening of the Achilles tendons; (6) bilateral pes cavus and flexion deformities of the toes; (7) multiple decubitus ulcers, and (8) mild bilateral congenital coxa valga.

On Nov. 9, he underwent bilateral lengthening of the Achilles tendons without anesthesia. Subsequently, he had a number of plastic operations for decubitus ulcers, a hemorrhoidectomy, and orthopedic operations.

On admission the patient was able to be up in a wheel chair but was unable to walk. He was started in December, 1948, on a program of gymnasium and mat work to build up the arm and upper-trunk muscles preparatory to crutch walking with braces. This program was interrupted repeatedly, however, because of operations. Long aluminum leg braces with a pelvic band were fitted, and the patient started on gait training in parallel bars and on crutch walking in August, 1949. In December, 1949, daily individual physical therapy for resistance exercises and neuromuscular reeducation of the trunk muscles was started. By May, 1950, he had improved sufficiently that he was able to walk with crutches and long leg braces without the pelvic band in a four-point gait without assistance for a distance of about 150 ft. (56 meters), but still required slight assistance with the swing-through gait on crutches. He then went home on a two-month furlough and resumed intensive treatment on July 5, 1950, which included intensive neuromuscular reeducation, gymnasium and mat work, and gait training. At this time it was noted that he had minimal voluntary contraction in the hip muscles. By October, 1950, there was noticeable return of sensation in some segments below the injury.

At the present time (June, 1951) the patient is in good health. He weighs 158 lb. (71.7 kg.), although he weighed 121 lb. (54.9 kg.) on admission to the hospital. He has no decubitus ulcers. He can now walk with long leg braces and crutches without assistance in a four-point gait for a distance of eight blocks, clearing easily at each step. He can swing-through on crutches and braces for over a block. He can walk on rough ground and grass and up and down steps, curbs, and ramps. He can at times walk the full length of the parallel bars with the left knee brace unlocked. The abdominal muscles, the lumbar sacrospinalis, and the quadratus lumborum are now good. He has voluntary contractions, with slight power and range of motion, in the psoas, adductors and tensor fasciae latae bilaterally. There is slight voluntary contraction in the quadriceps on each side. A very slight flicker response is noted in motions of the ankles and toes on voluntary effort with facilitation. He has slight voluntary contractions of the gluteus maximus and gluteus medius and the hamstring muscles bilaterally.

The muscular contractions in the hips and knees can be felt by the patient. He can also feel passive stretch of these muscles. Appreciation of position has returned to a moderate extent in the hips, more especially on the left, is considerably reduced in the left knee, but is absent in the right knee. Appreciation of vibration is present in both lower extremities, being greatly reduced below the knees. The patient now has normal touch and pain sensation through the second lumbar dermatome. He states that normal feeling in the region of the second lumbar dermatome has returned only in the last several months. Touch and pain sensations are both present but are slightly diminished in the third lumbar dermatome. There is very slight touch sensation in the fourth lumbar dermatome. No sensation is present in the fifth lumbar or the first sacral dermatome. On the other hand, the second sacral dermatome has almost normal sensation to both touch and pain. However, the sensation in this dermatome is peculiar in that the patient consistently localizes stimulation of the left leg to the right and vice versa. The third sacral dermatome has touch and pain sensation throughout but has spots of good sensation and areas of diminished sensation. The fourth sacral dermatome has practically normal sensation.

The very slight feeling of bladder fulness which he had on admission has gradually improved, so that he can now feel very well when his bladder is filling and therefore has time to go in his wheel chair a distance of over a block and reach the bathroom without wetting himself. The major return of bladder feeling has occurred during the past eight months. He has noted in the past month the development of slight ability to control urination by stopping the stream for a moment. He cannot initiate urination voluntarily but can help to start automatic contraction of the detrusor by pressure on the lower part of the abdomen. He now has some bowel sensation but no voluntary control. The patient is continuing intensive treatment and is showing steady improvement in motor and sensory function below the level of the spinal-cord injury.

Case 3.—A man aged 22 was injured in a cave-in and slate fall in a coal mine in Wyoming on Nov. 25, 1949. Immediately after the accident he had complete loss of voluntary motion and sensation from the middle of the chest down. After the injury, the left arm was numb and paralyzed, but this condition cleared in the course of a month. Laminectomy was not performed. There was no improvement in sensation or voluntary motion from the time of the injury until he was admitted to the Kabat-Kaiser Institute, in California, on July 21, 1950.

The essential findings on admission were (1) old comminuted fracture of the 11th thoracic vertebra, narrowing of the interspace between the 11th and the 10th thoracic vertebra, and fracture of the transverse process of the 11th thoracic vertebra; (2) normal sensation through the 4th thoracic and slight residual sensation in the 5th thoracic dermatome, with complete loss of sensation below this level; (3) complete paralysis below the 4th thoracic segment; (4) incontinence of bowel and bladder, no bowel or bladder sensation, and automatic reflex bladder, and (5) decubitus ulcers on the sacrum and heels.

Although voluntary contractions were not apparent in any of the muscles below the fifth thoracic segment when he attempted to move by himself, they were noted in the abdominal muscles and the quadratus lumborum during the original examination when mass-movement patterns of the upper and lower trunk were combined against resistance. It was also possible to demonstrate very weak voluntary contractions in muscles of the hip and knee with techniques of maximal facilitation.

While the patient without motor or sensory function below the fourth to the fifth thoracic segment may be able to ambulate on crutches with a three-point gait if he has two good arms and these are greatly developed, it is not possible for such a patient to walk effectively with a four-point gait with braces and crutches because of the complete absence of pelvic motion.

On Aug. 19, the patient was started on an intensive program of physical therapy, techniques of maximal facilitation being used to develop voluntary contraction of the muscles of the lower trunk, hips, and knees and of the intercostal muscles below the level of the spinal-cord injury. He was also given a program of gymnasium and mat work and was measured for long aluminum leg braces and started on attempts to walk in parallel bars and on crutches. By November, 1950, there was considerable improvement in the trunk muscles below the sensory level, and the patient was able to start walking with a four-point gait with long leg braces and crutches. The decubitus ulcers healed with conservative treatment. While he weighed 110 lb. (49.9 kg.) on arrival he now weighs 138 lb. (62.6 kg.).

At the present time (June, 1951) this patient is able to walk with long aluminum leg braces and crutches for about one-half block without assistance, using a four-point gait pattern. He can walk on rough ground and can go up and down the ramp, but has not been able to go up and down stairs or curbs. He is able to ambulate with a swing-through gait for a short distance on crutches and braces without assistance.

He now has fair power of voluntary contraction in the quadratus lumborum and the abdominal muscles and in the erectores spinae of the lower thoracic and lumbar region. Respiration is now good, and the intercostal muscles are definitely contracting below the sensory level. Slight voluntary contractions can be observed in the flexors and adductors of the hip, the tensor fasciae latae, and the quadriceps bilaterally. He has very slight flicker contractions in the muscles of the ankles and in the hip extensors. He has slight contractions in the knee flexors. He has no voluntary control of bladder or bowels.

There are normal sensation through the fourth thoracic and diminished sensation in the fifth thoracic dermatome. In the past two months there has been return of slight sensation below this level. He can feel contractions of muscles below the sensory level in the lower trunk and hips and to some extent in the thighs, but cannot feel stretch of the muscles. If the examiner rubs his leg, he can tell which side it is, but his localization is poor. For example, when the right thigh above the knee was rubbed, he thought the area was below the knee but localized the side correctly. He can tell roughly when the rubbing stops and starts. Temporal summation of sensory stimuli is essential for a response. He has markedly diminished sensation to pinprick in the legs, much as he has to rubbing. He does not perceive sharp pain but feels burning and localizes it in the correct leg, but not in the correct area. This vague sensation is present in all segments below the sensory level through the third sacral. During the past month he has noted a burning feeling in the bladder associated with filling.

SUMMARY

Patients who have had no functional recovery for a considerable period (in some cases many years) after apparently complete traumatic paraplegia have acquired voluntary motion below the level of the spinal-cord injury through intensive neuromuscular reeducation of a new type. Three cases are presented which illustrate a much larger experience.

These methods of neuromuscular reeducation produce maximal excitation in motor mechanisms in the central nervous system. The techniques can be applied for effective neuromuscular examination, as well as for treatment.

With this method of clinical muscle testing, it was possible to demonstrate in each of these cases a residual dormant motor connection through the injured area of the spinal cord to the lower segments. No evidence of residual voluntary motion below the level of the spinal-cord injury could be demonstrated in these cases by the usual methods of muscle testing. Intensive treatment has confirmed these observations as the patient gradually obtained voluntary contraction in muscles innervated by segments below the spinal-cord injury which had previously been completely paralyzed.

Gradual return of sensation below the previous sensory level associated with restoration of voluntary motion has been observed in some of these cases.

Clinical differentiation of complete from incomplete transection of the spinal cord is discussed.

Reasons for failure of intact nerve fibers in the region of the spinal-cord injury to resume function spontaneously are discussed.

LESIONS OF THE CENTRAL NERVOUS SYSTEM IN DISSEMINATED LUPUS ERYTHEMATOSUS

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THERE have been numerous reports of the manifestations of acute disseminated lupus erythematosus in the various organs of the body since Kaposi [1] first formulated the concept of the grave form with systemic disturbances. Neurologic signs and symptoms have been reported to occur in the course of the disease, but it has been indicated that the involvement of the central nervous system is not well understood and that the morbid anatomy of the nervous system in this illness requires much further investigation.[2]

Since 1930, autopsy has been performed in 18 cases of acute lupus erythematosus at the Presbyterian Hospital. The central nervous system was examined in six cases, and in three of these widespread lesions were found. It is the purpose of the present paper to report these cases in detail and to review the pertinent literature.

REPORT OF CASES

CASE 1.—L. C., an unmarried white woman aged 22, was admitted to the Presbyterian Hospital on April 4, 1945, with a history of nausea and vomiting daily and a nonpruritic rash on the dorsa of the hands, and the palms, forearms, and cheeks, and over the bridge of the nose for three weeks prior to admission. A two-month episode of "Sydenham's chorea" was reported to have occurred one year previously.

Physical Examination.—On admission the patient was acutely ill, with a temperature of 101 F. The blood pressure was 140 mm. systolic and 95 mm. diastolic. An erythematous rash was present on the dorsa of the hands, and the palms, forearms, cheeks, and bridge of the nose. The right eye was amblyopic, and the right optic disk was pale and surrounded by irregular red and white patches in the fundus. Tenderness and increased resistance were present in the right upper abdominal quadrant. The heart and lungs were normal.

Neurologic Examination.—No abnormalities were noted.

Laboratory Data.—The following significant findings were noted: slight anemia and leucopenia, hematuria, and albuminuria; a 4+ reaction in the cephalin flocculation test, and elevation of the erythrocyte sedimentation rate to 110 mm. per hour.

Course in the Hospital.—The temperature varied from 100 to 102 F. The patient gradually became irrational and uremic, with the blood urea nitrogen rising to 114 mg. per 100 cc. She died 12 days after admission.

From the Department of Neurology, Columbia University College of Physicians and Surgeons, and the Neurological Institute, Presbyterian Hospital.

1. Kaposi, M.: Neue Beiträge zur Kenntnis der Lupus erythematosus, Arch. Dermat. u. Syph. **4**:36-42, 1872.

2. (*a*) Libman, E.: Some Aspects of Libman-Sacks Disease, J. Mt. Sinai Hosp. **9**:621-630, 1942. (*b*) Moschcowitz, E.: Libman-Sacks' Disease, in Biology of Disease, New York, Grune & Stratton, Inc., 1948, pp. 36-47. (*c*) Russell, P. W.; Haserick, J. R., and Zucker, E. M.: Epilepsy in Systemic Lupus Erythematosus, A. M. A. Arch. Int. Med. **88**:78-92, 1951.

Autopsy.—A complete necropsy was performed, and the following anatomical diagnoses were made: lupus erythematosus disseminata; verrucous endocarditis of the mitral and tricuspid valves and the right auricle (Libman-Sacks type); endarteritis (generalized) due to lupus erythematosus; encephalomalacia (multiple, old); infarcts of the kidneys; glomerulosclerosis; splenomegaly (with periarterial fibrosis of the spleen and acute fibrinous perisplenic peritonitis); coronary arteriosclerosis with myocardial fibrosis; acute diphtheritic pharyngitis, laryngitis, and tracheobronchitis; bilateral bronchopneumonia, and fatty liver.

Central Nervous System: Gross examination: Multiple areas of focal atrophy of the cerebral cortex were present, primarily involving the convexity gyri. Some of these lesions extended into the superficial portion of the subcortical white matter, and in a few areas tiny cysts were present. There was marked congestion of the leptomeningeal vessels. The cerebellum, brain stem, and spinal cord appeared grossly normal.

Histologic Examination: There were many focal areas of encephalomalacia corresponding to the atrophic regions noted grossly in the frontal, temporal, and parietal gyri. The cortex

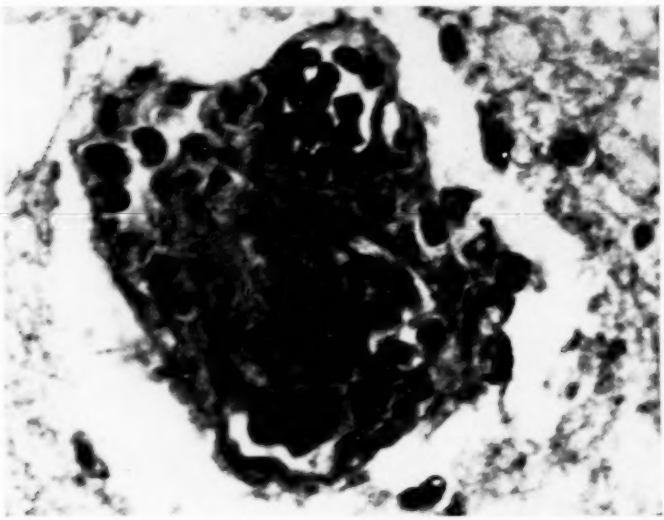

Fig. 1 (Case 1).—Arteriole in right frontal cortex, showing pronounced "fibrinoid" degeneration with occlusion of the lumen and a small amount of perivascular infiltration. Hematoxylin-eosin stain; × 450.

at the sulcal margins was degenerated and rarefied, owing to extreme loss of neurons and glia. In these areas there was a meshwork of glial fibers and blood vessels, surrounded by large mononuclear phagocytes. A rich astrocytosis was present at the margins of the degenerated zones. Small numbers of lymphocytes and large mononuclear phagocytes were present in the subarachnoid spaces and the pia. A branch of the left middle cerebral artery was thrombosed, and the walls of this vessel showed intimal proliferation and fraying of the intimal elastic fibers. Many other smaller arteries and arterioles throughout the parenchyma and meninges showed the following changes, in varying degrees: intimal proliferation; irregular patches of eosinophilic material ("fibrinoid"), extending from the adventitia through the media to the intima, and often into the lumen; congestion with erythrocytes, and perivascular infiltration with lymphocytes and occasional polymorphonuclear leucocytes (Fig. 1).

No abnormalities were noted in the brain stem or spinal cord.

Case 2.—B. B., a housewife aged 48, was admitted to the Neurological Institute on Feb. 19, 1947, in a comatose state following a grand mal seizure.

She had a well-documented 14-year history of Raynaud's syndrome involving all four extremities. Clinical examination in 1944 showed a blood pressure of 117 mm. systolic and 70 mm. diastolic, questionable capillary hemorrhages or dilatations in the skin, and blanching of the fingers and toes on exposure to mild cold. She complained of "dizzy spells" at that time. Bilateral dorsal sympathectomy was performed in 1945, with some relief of symptoms. In 1946 intermittent claudication appeared in both lower extremities. Shortly after this, in October, 1946, lumbar sympathectomy was performed on the left side. A grand mal seizure occurred in the immediate postoperative period. Several months later, three more generalized seizures occurred, each preceded by a "raving, confused, agitated" state. The patient became depressed and bedridden and complained of severe pain in the fingers. Nausea and vomiting occurred frequently. On the day of admission she had a severe generalized convulsion, followed by coma.

Physical Examination.—The patient was comatose, cyanotic, and dyspneic. The temperature was 99.6 F.; the pulse rate, 100 per minute, and the respiration rate, 26 per minute. The blood pressure was 126 mm. systolic and 82 mm. diastolic. Moist rales were heard at the base of the left lung. The heart was normal. The liver was palpable 4 in. (10 cm.) below the costal margin. A brown pigmentation was present in the skin of the abdomen, hands, and arms. The skin of the fingers and forearms was smooth and glassy in appearance, and the tips of the fingers were markedly atrophic and clubbed.

Neurologic Examination.—A complete examination could not be performed. The right leg was spastic, and right-sided hyperreflexia, without pathological toe reflexes, was present. The left extremities were flaccid. Laboratory studies could not be made. The patient died 13 hours after admission.

Autopsy.—A complete necropsy was performed, and the following anatomical diagnoses were listed: endarteritis of the small arteries of the cerebrum (acute lupus erythematosus type); sclerosis of small arteries in the kidneys, lungs, liver, uterus, and brain (chronic lupus erythematosus type); encephalomalacia (multiple, recent and old); arteriolar nephrosclerosis, atypical; acute verrucous endocarditis of the mitral and aortic valves; mural thrombus in the heart (right ventricle); edema and fibrosis of the myocardium (mild); focal necrosis of the liver, and bilateral pneumonia.

Central Nervous System: Gross examination: There was mild, generalized atrophy of the frontal gyri bilaterally. A great number of small, irregular, rounded depressions were present over the entire cortex. Mild congestion of the leptomeningeal blood vessels was present. The cerebellum, brain stem, and spinal cord were grossly normal.

Histologic Examination: The atrophic areas noted grossly contained lesions in the blood vessels with surrounding acute and chronic encephalomalacia in the gray and white matter. There were wide areas of degeneration and edema in the gyri on either side of many sulci. In these areas the neurons in all layers exhibited ischemic necrosis. There were many regions with diffuse loss of neurons and glia and infiltration with polymorphonuclear leucocytes. Astrocytosis was often present in nodular formations or around small cystic zones (Fig. 2*A*). The vascular lesions were both acute and chronic. Masses of homogeneous, eosinophilic, "fibrinoid" material were present in the walls of many small arteries and arterioles, involving principally the intima (Fig. 2*B*). The lumen was often occluded by extrusion of this "fibrinoid" material or by endothelial and subendothelial proliferations. There were severe degeneration of the elastica and extensive thickening and fibrosis in all layers of many vessels. Perivascular infiltration with polymorphonuclear leucocytes and large mononuclear cells was common. Channelized old thrombi were noted in several medium-sized leptomeningeal arteries.

The hippocampal gyrus, corpus striatum, thalamus, cerebellum, midbrain, and pons had diffuse lesions similar to, but much milder than, those noted in the cerebral cortex. The spinal cord was normal throughout.

Case 3.—P. S., a graduate nurse and housewife aged 23, was admitted to Presbyterian Hospital on July 30, 1949, with high fever, joint pains, anemia, and delirium.

In 1946, when a student nurse, she noticed a rash over her eyes and forehead, which was believed to be due to sensitivity to adhesive plaster. In 1947, she was admitted to the hospital with an illness characterized by arthritis, fever, generalized lymphadenopathy, severe asthenia, and a positive cephalin flocculation reaction. A diagnosis of atypical infectious mononucleosis

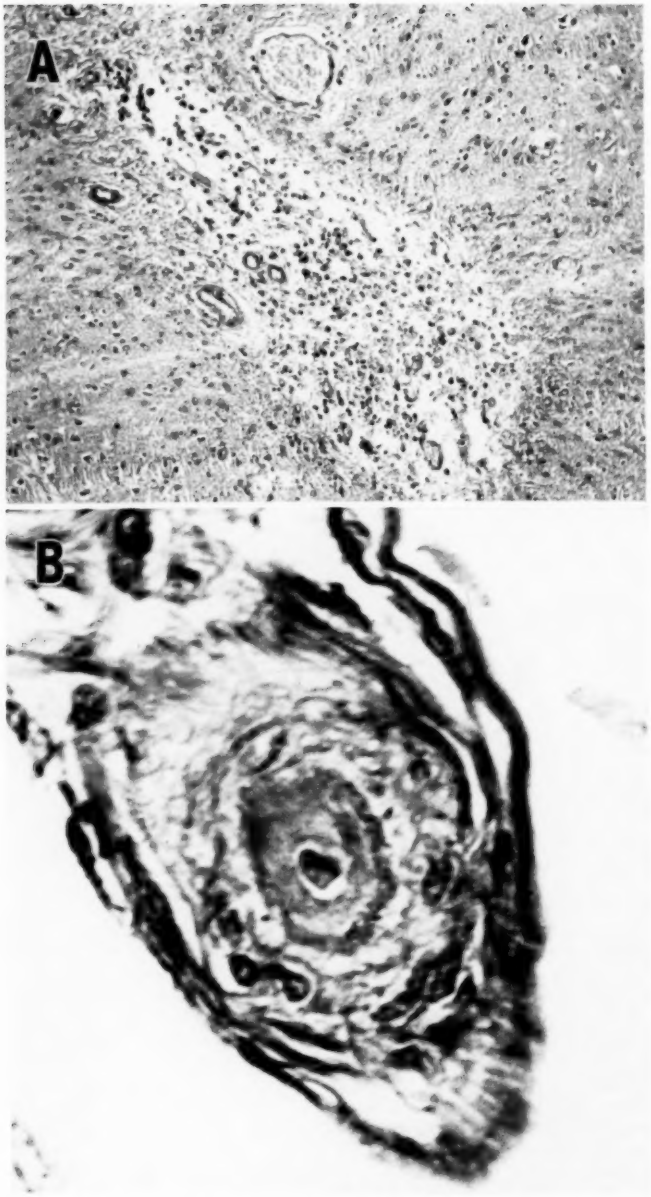

Fig. 2 (Case 2).—*A*, small focal area of encephalomalacia in right frontal region. Phosphotungstic-acid-hematoxylin stain; × 150.

B, arteriole in leptomeninges, right frontal region, showing endothelial and subendothelial proliferation. A heavy ring of "fibrinoid" material has occluded the lumen almost completely. Hematoxylin-eosin stain; × 450.

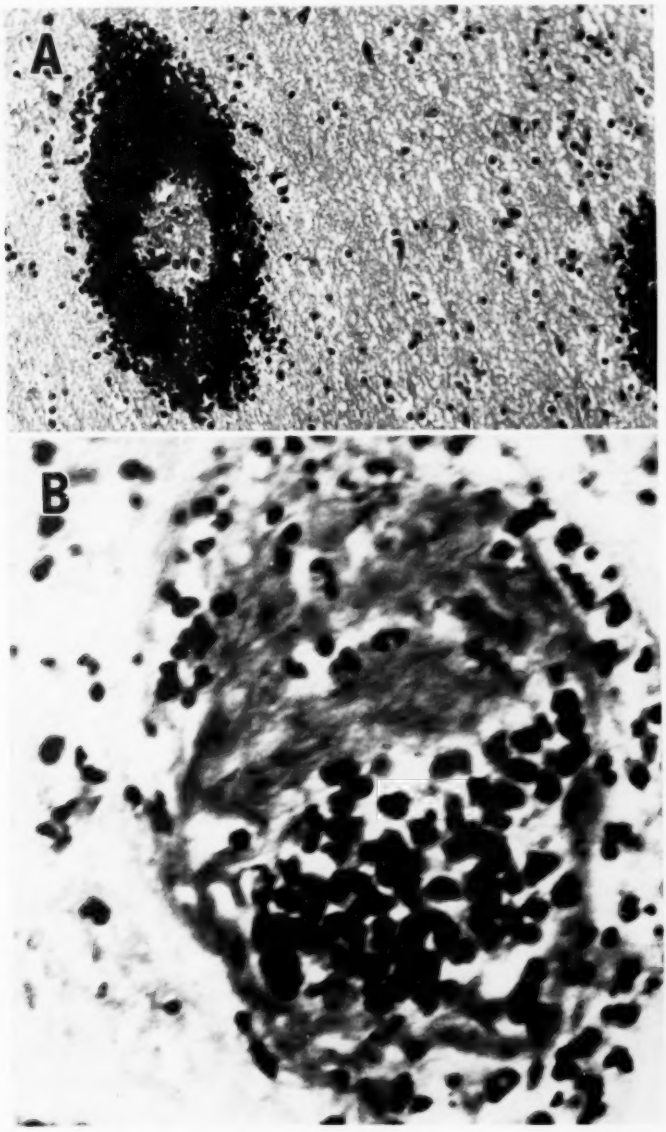

Fig. 3 (Case 3).—*A*, ring hemorrhage in cerebral cortex of the right inferior frontal gyrus. Hematoxylin-eosin stain; × 150.

B, arteriole in the right inferior frontal gyrus, showing "fibrinoid" degeneration and necrosis in the vessel wall and perivascular infiltration with polymorphonuclear leucocytes. Hematoxylin-eosin stain; × 450.

was made, and during the following year these symptoms and signs persisted. In 1948 her condition improved and she married. Ten months prior to admission she became pregnant, and during the latter half of the pregnancy the joint pains became severer. After the delivery the arthritis, fever, and anemia became worse, and disorientation and delirium appeared.

Physical Examination.—The patient was acutely ill, lethargic, and disoriented. The temperature was 101.6 F.; the pulse rate, 116 per minute; the respiration rate, 24 per minute, and the blood pressure, 94 mm. systolic and 60 mm. diastolic. Significant physical findings included a pericardial friction rub, gallop rhythm, splenomegaly, and fusiform swellings of the interphalangeal joints.

Neurologic Examination.—The results of the neurologic examination were entirely normal. The mental status showed variations in the level of awareness. She was frequently in a state of delirium with disorientation.

Laboratory Data.—The complete blood count was normal. Hematuria and albuminuria were present. The erythrocyte sedimentation rate was 22 mm. in one hour, and the cephalin flocculation reaction was 4+. An electrocardiogram showed nodal tachycardia and a prolonged conduction rate. A roentgenogram of the chest revealed cardiac enlargement and pleuritic involvement.

Course in the Hospital.—On Aug. 1, treatment with corticotropin was started in doses varying from 60 to 100 mg. daily. The arthritic symptoms and friction rub rapidly disappeared; the temperature and the pulse rate became normal, and the spleen became smaller. However, after 16 days of treatment she was allowed out of bed, when she had sudden severe chest pain, dyspnea, tachycardia, and fever. It was thought that a pulmonary infarction had occurred. She died shortly thereafter.

Autopsy.—A complete necropsy was performed, and the following anatomical diagnoses were listed: acute disseminated lupus erythematosus; verrucous endocarditis; acute fibrinous pericarditis with effusion; myocardial hypertrophy and dilatation; acute pneumonitis; pulmonary emboli with infarction; adrenal hyperplasia (bilateral); hemorrhagic encephalomalacia (right frontal, temporal, and parietal lobes); thrombi in the perivesicular venous plexus, and acute and chronic lymphadenitis (retroperitoneal lymph nodes).

Central Nervous System: Gross examination: Small hemorrhagic lesions were seen chiefly in the cortex of the right frontal, parietal, and temporal lobes. The cerebellum, brain stem, and spinal cord appeared to be normal.

Histologic examination: The lesions in the frontal, parietal, and temporal gyri were studied. Many small and a few large hemorrhagic areas were noted; they involved chiefly the cortex, but extended into subcortical white matter. Fresh ring hemorrhages around small arteries and arterioles were common (Fig. 3*A*). The vessels were involved by endothelial hyperplasia, necrosis of adventitia and media, and perivascular and mural infiltration with polymorphonuclear leucocytes (Fig. 3*B*). Occasional intramural masses of eosinophilic "fibrinoid" material were present. The intervening neural tissue was edematous and infiltrated with polymorphonuclear leucocytes, and widespread ischemic degeneration and loss of neurons were present. There were no vascular changes in the basal ganglia, but occasional neurons in the caudate nucleus and putamen were deeply stained and shrunken and had pyknotic nuclei. The midbrain and cerebellum were normal. Several small recent hemorrhages were noted in the floor of the fourth ventricle, but otherwise no significant changes were noted in the lower part of the brain stem or the spinal cord.

COMMENT

These observations indicate that extensive focal and diffuse lesions of the central nervous system may occur in disseminated lupus erythematosus. That neurologic signs and symptoms (aside from those of delirium) may be absent despite these widespread lesions is illustrated by two of the three cases.

The diagnosis of acute disseminated lupus erythematosus is usually made clinically when an acute febrile illness is associated with polyarthritis, a characteristic "butterfly" cutaneous lesion of the face, and signs of pulmonary, cardiac, and renal

involvement. Splenomegaly is frequent. Laboratory findings include a greatly elevated erythrocyte sedimentation rate and serum globulin; leucopenia, and occasional thrombocytopenia. Recently, studies of the lupus erythematosus cell in the bone marrow and the plasma lupus-erythematosus phenomenon have established more confirmatory diagnostic methods.[3]

Neurologic symptoms have been reported to occur during the course of this illness; these have included focal and generalized seizures in the absence of azotemia,[4] transient cranial nerve paralysis,[5] hemiparesis, and aphasia.[6] Keil [7] found a first-zone colloidal gold curve in the cerebrospinal fluid in one case. "Papilledema" is an occasional finding but is usually regarded to be due to a local retinal lesion and occurs in the absence of increased cerebrospinal fluid pressure.[8] Manifestations of an acute toxic psychosis or delirium are frequently noted, especially terminally, in association with high fever or uremia, and do not necessarily indicate the presence of focal cerebral lesions.

Several authors have described lesions of the central nervous system, but the reports have been brief and often vague. These lesions have included subarachnoid and extra-arachnoid hemorrhage,[9] cerebral endarteritis and encephalomalacia,[10] and intracerebral gross and petechial hemorrhages.[4] Sedgwick and Von Hagen [11] reported five cases with neurologic symptoms; but in none was there pathologic examination of the nervous system, and the confirmation of the diagnosis of lupus erythematosus was by biopsy of facial skin.

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3. Hargraves, M. M.; Richmond, H., and Morton, R.: Presentation of Two Bone Marrow Elements: "Tart Cell" and "L.E." Cell, Proc. Staff Meet., Mayo Clin. **23**:25-27, 1948. Lee, S. L.; Michael, S. R., and Vural, I. L.: The L.E. (Lupus Erythematosus) Cell: Clinical and Chemical Studies, Am. J. Med. **10**:446-451, 1951.

4. (*a*) Russell, Haserick, and Zucker.[2c] (*b*) Jarcho, S.: Lupus Erythematosus Associated with Visceral Vascular Lesions, Bull. Johns Hopkins Hosp. **59**:262-274, 1936. (*c*) Klemperer, P.; Pollack, A. D., and Baehr, G.: Pathology of Disseminated Lupus Erythematosus, Arch. Path. **32**:569-631, 1941. (*d*) Baldwin, G. B.: Acute Disseminated Lupus Erythematosus, with Report of Fatal Case, M. J. Australia **2**:11-15, 1945. (*e*) Montgomery, H., and McCreight, W. G.: Disseminate Lupus Erythematosus, Arch Dermat. & Syph. **60**:356-372, 1949. (*f*) Tumulty, P. A., and Harvey, A. M.: Clinical Course of Disseminated Lupus Erythematosus: An Evaluation of Osler's Contributions, Bull. Johns Hopkins Hosp. **85**:47-73, 1949.

5. Friedberg, C. K.; Gross, L., and Wallach, K.: Non-Bacterial Thrombotic Endocarditis Associated with Prolonged Fever, Arthritis, Inflammation of Serous and Widespread Vascular Lesions, Arch. Int. Med. **58**:662-684, 1936.

6. (*a*) Klemperer, Pollack, and Baehr.[4c] (*b*) Montgomery and McCreight.[4e] (*c*) Tumulty and Harvey.[4f] (*d*) Daly, D.: Central Nervous System in Acute Disseminate Lupus Erythematosus, J. Nerv. & Ment. Dis. **102**:461-465, 1945.

7. Keil, H.: Dermatomyositis and Systemic Lupus Erythematosus, Arch. Int. Med. **66**: 339-383, 1940.

8. Maumenee, A. E.: Retinal Lesions in Lupus Erythematosus, Am. J. Ophth. **23**:971-981, 1940.

9. Tremaine, M. J.: Subacute Pick's Disease with Polyarthritis and Glomerulonephritis, New England J. Med. **211**:754-756, 1934.

10. Jarcho.[4b] Montgomery and McCreight.[4e] Friedberg, Gross, and Wallach.[5] Daly.[6d] Cluxton, H. E., and Krause, L. A. M.: Acute Lupus Erythematosus Disseminatus, Ann. Int. Med. **19**:843-872, 1943.

11. Sedgwick, R. P., and Von Hagen, K. O.: Neurological Manifestations of Lupus Erythematosus and Periarteritis Nodosa, Bull. Los Angeles Neurol. Soc. **13**:129-142, 1948.

The lesions described in the central nervous system are due to the primary involvement of the smaller vessels of the arterial tree, a disseminated endarteritis. According to Klemperer,[12] the basic lesion in acute lupus erythematosus is one of "fibrinoid degeneration" of collagen, with a predilection for the small arteries and arterioles. The sequence of events in this type of necrotizing arteritis has been described by Klemperer, Pollack, and Baehr[13]: (1) a "fibrinoid" change in the collagenous framework in the intima and adventitia and between the muscles of the media, giving the appearance of fibrinoid deposition; (2) a fusion of "fibrinoid" material in all layers to form conglomerate masses of refractile tissue, intensely eosinophilic; (3) destruction of muscle and elastic tissue elements and accumulations of masses of necrotic connective tissue and basophilic fragments of nuclei, and (4) occlusion of the vessel by these masses. Endothelial proliferations may occur, and thrombus formations then often occlude the involved vessels. These changes were present in the three cases described in the present report. Acute vascular lesions were present in all three cases, and more chronic lesions in Case 2. In addition to the basic lesions, small and large hemorrhages, usually perivascular, were present throughout the cortex in Case 3.

It is noted in the present series that the vascular lesions of this disease in the central nervous system have a predilection for the gray matter of the cerebral cortex, particularly the perisulcal areas. The white matter is much less frequently implicated, and then usually by extension. This difference may only reflect the greater arterial supply of the gray matter. The frequency of convulsive seizures as a neurologic complication may be related to this cortical predilection.

Thus, in all instances the lesions in the brain parenchyma were secondary to the involved vascular supply, and changes of focal or disseminated acute and chronic encephalomalacia, often with small cyst formations, were present. Even though the vascular supply of the basal ganglia, diencephalon, and brain stem is extensive, lesions in these subcortical areas were noted only infrequently, and were then only of mild degree.

The close relationship between acute disseminated lupus erythematosus and scleroderma is indicated by Case 2 of this series. The vascular lesions showed the typical features of acute lupus and transitional stages between the acute and the healed or chronic phase. The clinical picture was not clearly that of acute disseminated lupus erythematosus, but resembled that of Raynaud's syndrome, suggesting a sclerodermatous basis. There is much discussion concerning this relationship,[14] and it is thought that these two illnesses may represent different phases of a similar pathologic process.

The presence of involvement of the central nervous system in acute disseminated lupus erythematosus is regarded as significant also because of recent problems developing in the course of the new treatment of this illness by induction of the hyperadrenocortical state by cortisone and the pituitary adrenocorticotropic hormone (corticotropin). This treatment seems to have a beneficial effect, even

12. Klemperer, P.: Pathogenesis of Lupus Erythematosus and Allied Conditions, Ann. Int. Med. **28**:1-12, 1948.

13. Klemperer, P.; Pollack, A. D., and Baehr, G.: Diffuse Collagen Disease: Acute Disseminated Lupus Erythematosus and Diffuse Scleroderma, J. A. M. A. **19**:331-332, 1942.

14. Pollack, A. D.: Visceral and Vascular Lesions in Scleroderma, Arch. Path. **29**:859-861, 1940.

though transient.[15] A total of 15 patients were treated by the investigators cited. In general, the temperature fell, and the signs of involvement of the joint, heart, lung, liver, spleen, kidney, and skin became less. However, the clinical course in two cases was punctuated by new signs of acute involvement of the central nervous system: subarachnoid hemorrhage in one case[15a] and epileptic seizures in another.[15b] In neither of these cases were neurologic signs present prior to the treatment. By contrast, Russell, Haserick, and Zucker[2c] observed that cortisone and corticotropin treatment produced improvement in epileptiform seizures related to lupus erythematosus. The spontaneous occurrence of seizures in untreated patients makes the appearance of attacks during treatment difficult to interpret. Also, the nature of the effect of the hyperadrenocortical state on these vascular lesions has not yet been determined.

SUMMARY

Vascular lesions of acute disseminated lupus erythematosus in the central nervous system are described in three cases. The picture is that of focal or disseminated encephalomalacia primarily involving the gray matter of the cerebral cortex and secondary to the specific endarteritis. In two cases neurologic signs and symptoms were absent despite extensive cerebral lesions. In the third case, generalized seizures and coma developed, but the involvement of the brain was too widespread to permit the association of any particular focal lesions with the symptoms.

The pertinent literature is reviewed, particularly that concerning previous reports of neurologic phenomena. The significance of these findings is discussed in relation to the recent treatment of this condition with cortisone and corticotropin (pituitary adrenocorticotropic hormone).

Dr. Abner Wolf advised and assisted in the preparation of the paper.

15. (*a*) Plotz, C. M.; Blunt, J. W., Jr., and Ragan, C.: Effect of Pituitary Adrenocorticotropic Hormone (ACTH) on Disseminated Lupus Erythematosus, Arch. Dermat. & Syph. **61**:913-918, 1950. (b) Soffer, L. J.; Levitt, M. F., and Baehr, G.: Use of Cortisone and Adrenocorticotropic Hormone in Acute Disseminated Lupus Erythematosus, Arch. Int. Med. **86**:558-564, 1950. (*c*) Brunsting, L. A.; Slocumb, C. H., and Didcoct, J. W.: Effects of Cortisone on Acute Disseminated Lupus Erythematosus, Proc. Staff Meet., Mayo Clin. **25**:479-482, 1950.

EFFECT OF PHLORHIZIN ON EXCRETION OF INORGANIC PHOSPHATE IN PSYCHOTIC PATIENTS

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FOR OVER a century increased urinary excretion of phosphate has been reported in some patients with manic-depressive, involutional, or schizophrenic psychoses; the mechanism of this change has not been established. Increases in reabsorption of phosphate by renal tubules following injection of phlorhizin has been described by Pitts and Alexander.[1] It was considered desirable to study the effects of phlorhizin in psychotic patients in order to ascertain whether the renal tubular reabsorption of phosphate is affected in a normal fashion by that substance.

MATERIAL AND METHODS

Sixteen experiments were performed on nine patients with manic-depressive, involutional, or schizophrenic psychoses. Six of the patients were women; the ages of the entire series ranged from 21 to 64. An additional experiment was performed on one of the schizophrenic patients when his phosphate excretion rose markedly at the onset of a viral infection.

The patients were studied while at rest and in the postabortive state. After control samples were obtained, 1 gm. of phlorhizin, dissolved in 5 ml. of propylene glycol, was injected intramuscularly; an additional gram was injected an hour later. The excretion of urinary phosphate was studied during a period of an hour and a half prior to the injection of phlorhizin and for five hours after it; the method of Fiske and Subbarow [2] was used. In five additional experiments made on other patients, the urinary phosphate excretion was studied before and for four hours after the injection of sodium chloride solution. Blood inorganic phosphate levels were measured before and again three and five hours after injection of phlorhizin in 10 experiments, the method of Fiske and Subbarow [2] being used. Urinary acid phosphatase activity was measured before and after injection of phlorhizin in all experiments by means of a method described elsewhere [3]; the method of Seligman and associates [4] was used to estimate the acid and alkaline phosphatase activities of blood serum before and again three and five hours after injection of phlorhizin in eight experiments.

From the Laboratories of Clinical Physiology, McLean Hospital, Waverley, Mass., and the Department of Medicine, Harvard Medical School.

1. Pitts, R. F., and Alexander, R. S.: The Renal Reabsorptive Mechanism for Inorganic Phosphate in Normal and Acidotic Dogs, Am. J. Physiol. **142**:648, 1944.

2. Fiske, C. H., and Subbarow, Y.: The Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375, 1925.

3. Altschule, M. D.; Parkhurst, B. H., and Zager, G. R.: Measurement of Acid Phosphatase Activity of Urine, Am. J. Clin. Path. **21**: 480, 1951

4. Seligman, A. M.; Chauncey, H. H.; Nachlas, M. M.; Manheimer, L. H., and Ravin, H. A.: The Colorimetric Determination of Phosphatase in Human Serum, J. Biol. Chem. **190**:7, 1951.

OBSERVATIONS

The urinary output of inorganic phosphate in the period preceding the injection of phlorhizin ranged from 11.8 to 53.5 mg. per hour (Chart 1). The excretion

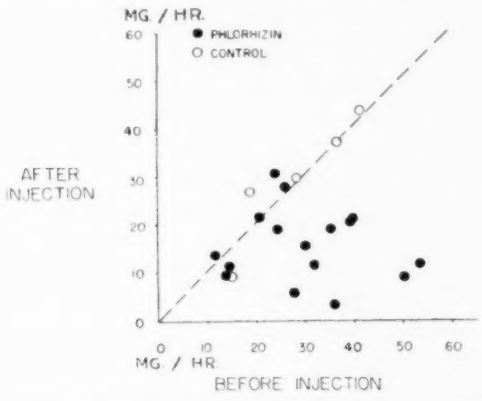

Chart 1.—Urinary phosphate excretion, expressed in milligrams per hour, before and after injection of phlorhizin (black dots); points to the right of the diagonal line show decreases. Open circles indicate studies made before and after injection of sodium chloride solution.

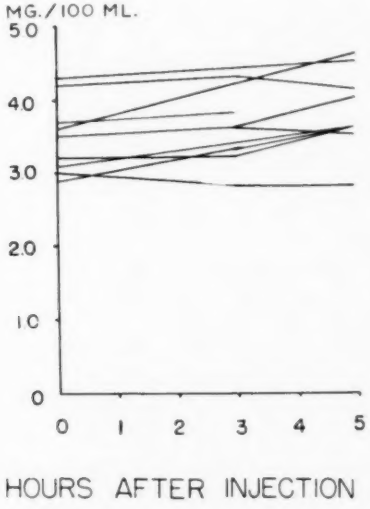

Chart 2.—Effect of phlorhizin on serum inorganic phosphate concentration, expressed in milligrams per 100 ml.

of inorganic phosphate after the injection of phlorhizin was between 3.4 and 30.8 mg. per hour (Chart 1). Comparison of outputs prior to and during the periods of the action of phlorhizin showed that increases in outputs of phosphate of between

1.1 and 6.8 mg. per hour occurred in four instances; decreases of between 3.0 and 5.5 mg. per hour were observed in three other experiments. In all the remaining experiments the average excretion of phosphate fell by more than 16 mg. per hour. When decreases occurred, they were evident in the first sample of urine, collected usually one hour after the first injection of phlorhizin. In the patient studied at the onset of a viral infection, when his urinary phosphate excretion was 109.5 mg. per hour, injection of phlorhizin was followed by a fall in output to 12.9 mg. per hour. Changes in excretion of phosphate over a period of four hours following the injection of sodium chloride solution were negligible (Chart 1).

The blood inorganic phosphate level showed increases of between 0.4 and 1.0 mg. per 10 ml. in five experiments (Chart 2); the average change in this group was 0.6 mg. per 100 ml. In five other experiments the maximal changes were within ± 0.2 mg. per 100 ml. of the control values.

Serum acid and alkaline phosphatase activities and urinary acid phosphatase activity showed no consistent changes during the experiments.

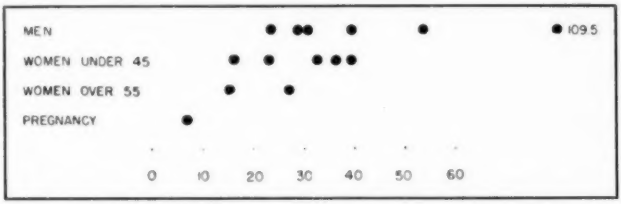

Chart 3.—Average fasting morning urinary phosphate output, expressed in milligrams per hour, in psychotic patients.

COMMENT

The hourly excretion of phosphate during control periods in the patients studied here was normal or high; the tendency toward increased excretion of phosphate was exhibited even in a pregnant psychotic patient, whose fasting output was 7 mg. per hour (Chart 3). These findings have no definable significance, however, in the absence of balance studies.

The results of the present study show that the effect of phlorhizin in psychotic patients is to decrease urinary output of phosphate; these findings are similar to those reported in the past by Pitts and Alexander.[1] Of particular interest are the observations made on one patient studied at the onset of a febrile illness: His phosphate excretion was increased to a degree similar to that observed by Pincus and associates[5] in psychotic patients given corticotropin (ACTH); after injection of phlorhizin his urinary phosphate excretion showed a pronounced decrease.

Renal vascular dynamics are not affected by phlorhizin; the action of that substance is to change renal tubular function. Although the observed decrease in phosphate excretion in all the patients occurred simultaneously with the inhibition of reabsorption of glucose, there was no gross correlation between these two effects

5. Pincus, G.; Hoagland, H.; Freeman, H.; Elmadjian, F., and Romanoff, L. P.: A Study of Pituitary-Adrenocortical Function in Normal and Psychotic Men, Psychosom. Med. **11**:74, 1949.

of the drug: Significant decreases in phosphate excretion occurred in 10 of 17 experiments, while approximately the same amount of sugar was excreted in all 17 experiments.

Failure of phlorhizin to depress phosphate excretion in some experiments may have been due to several factors. Loss of glucose from the body initiates gluconeogenesis, and phosphate is liberated with nitrogen in a fixed ratio.[6] When protein is broken down, increased amounts of phosphate are presented to the renal tubules for reabsorption, and therefore more phosphate may escape reabsorption and be excreted in the urine; the serum inorganic level rose in 5 of 10 experiments in the present study. The action of still another factor is suggested by the fact that decreases in blood eosinophile counts of more than 50% were found four hours after the injection of phlorhizin in all but three experiments, showing that activation of the adrenal cortex had occurred; one effect of the injection of corticotropin in psychotic subjects is a marked increase in the urinary excretion of phosphate.[5]

The excretion of phosphate was studied for several additional hours in two patients, and decreases still were found seven hours after the first injection of phlorhizin. The retention of phosphate so induced had no discernible effect on the mental state of any of the patients.

It is probable that abnormal phosphaturia encountered in some psychotic patients is the result of several factors: One probably is increased breakdown of body proteins associated with gluconeogenesis due to stress. Another possible factor is suggested by the results of the present study, that is, competition by various components of the glomerular filtrate for reabsorption by renal tubules. Increases in blood levels of some substances due to metabolic changes may give rise to decreased reabsorption of other substance by the renal tubules. The tendency of many psychotic patients to exhibit hyperglycemia and hyperketonemia under some circumstances is well known. This discussion is not intended to suggest that increases in blood glucose or ketones are the only important factors in this respect; other metabolites also may be implicated. In addition, other factors whose nature is as yet unknown may be responsible in part for abnormal phosphaturia of mental disease. On the other hand, a tendency toward increased phosphaturia may be masked by the failure to eat exhibited by psychotic patients or by their maintenance on institutional diets low in protein.

SUMMARY AND CONCLUSIONS

Injection of phlorhizin usually results in decreased urinary output of inorganic phosphate in psychotic patients whose phosphate excretion is normal or increased.

The excessive phosphaturia that may occur in mental disorders is consequent to several factors, one of which appears to be competition by various metabolites for renal tubular reabsorption.

6. Kastler, A. O.: The Influence of Phlorhizin upon Inorganic Metabolism, J. Biol. Chem. **76**:643, 1928.

ACUTE SUBDURAL SPINAL ABSCESS

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A SUBDURAL spinal abscess is a collection of pus, with associated inflammatory phenomena, contained exclusively between the dura and the spinal cord. Although extradural abscess of the spinal canal is rare, purulent collections limited to the subdural space are virtually unknown. A recent successfully treated acute subdural spinal abscess prompted a careful review of the literature to determine the incidence of this unusual condition.

The first case of this kind to be reported in the English literature was that of Bennett and Keegan,[1] in 1927. Since that time five additional cases[2] have been reported. Because of the rarity of this lesion, it appeared worth while to tabulate the clinical features of these six cases and to compare them with the findings in the present case.

REPORT OF A CASE

History.—F. L., a married white woman aged 30, entered the orthopedic service of Mount Sinai Hospital on Dec. 11, 1948, complaining of excruciating low-back pain. The onset of her illness began nine days prior to her admission. On that morning she awoke with transient pain in the back of her neck. Later that day she had onset of low-back pain, which was constant and was not affected by change in body position. At times she felt a "kinking" sensation in the left thigh. Each day the back pain grew progressively worse, and three days prior to her admission urinary retention developed.

The history revealed that the patient had fallen from a horse at the age of 20, but this had resulted in no permanent disability. Specific questioning of the patient after operation disclosed that about two weeks before the onset of her back pain she had picked at a furuncle in her nose until it bled.

Physical Examination.—The patient was a well-developed and well-nourished woman who appeared acutely ill, and in severe pain. The pulse rate was 100 per minute; the respiration rate, 20 per minute; the oral temperature, 101.8 F; the blood pressure, 112/0. The general physical examination, including a pelvic examination, revealed nothing significant. Examination of the back revealed exquisite tenderness over the lumbosacral and sacral regions of the spine, with

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2. (*a*) Arnett, J. H.: Meningococcic (Later Also Staphylococcic) Meningitis, Low Spinal Subarachnoid Block, Abscess, Laminectomy, Recovery, M. Clin. North America **13**:1051 (Jan.) 1930. (*b*) Chi, C. Y.: Intraspinal Subdural Abscess, Chinese M. J. **50**:921 (July) 1936. (*c*) Abbott, K. H.: Acute Subdural Spinal Abscess, Bull. Los Angeles Neurol. Soc. **5**:227 (Dec.) 1940. (*d*) Freedman, H., and Alpers, B. J.: Spinal Subdural Abscess, Arch. Neurol. & Psychiat. **60**:49 (July) 1948.

severe spasm of the paravertebral muscles. The patient held her back in extreme extension, and attempts at flexion of the spine resulted in severe pain. Aside from a distended bladder, due to urinary retention, the neurological examination gave essentially normal findings. There was no motor weakness; all reflexes were normal, and there were no disturbances of sensation.

Laboratory Data.—The red blood cell count was 3,100,000 per cubic millimeter. The hemoglobin was 10.5 gm. per 100 cc. The white blood cell count was 14,400 per cubic millimeter, with a normal differential count. The sedimentation rate was 47 mm. in one hour. The urine was normal. The Kline reaction of the blood was negative.

A roentgenogram of the chest showed nothing abnormal. The roentgenogram of the lumbar portion of the spine was normal except for narrowing of the fifth intervertebral space.

Hospital Course.—Treatment on admission consisted of traction to both legs, hot compresses to the back, intravenous administration of fluids, and sedation and analgesia. For the first three hospital days the patient had a continuous fever, the temperature ranging from 100 to 102 F. (oral); and her symptoms continued unabated. Neurosurgical consultation was called on the third hospital day; at that time a lumbar puncture was attempted, but only a few drops of yellow-tinged fluid were obtained. Because of the patient's extreme pain, it was necessary to perform a second lumbar puncture the next day with the patient under thiopental-sodium anesthesia. This puncture produced a few centimeters of creamy pus, which on smear revealed Gram-positive cocci. Subsequent culture isolated the Staphylococcus aureus (Micrococcus pyogenes var. aureus).

Surgical Procedure.—With a presumptive diagnosis of localized intraspinal abscess, the patient was prepared for immediate operation. Penicillin (300,000 units) was administered, and a transfusion was started. A complete laminectomy of the second, third, fourth, and fifth lumbar vertebrae and the first sacral vertebra was performed. The extradural tissues and ligamentum flavum were found to be very adherent and tough. The dura appeared tremendously thickened, but no extradural suppuration was seen. Needle aspiration through the dura brought forth pus, and when the dura was opened several pockets of pus were found, extending from the second lumbar to the first sacral segment. The purulent material was carefully aspirated with suction. The dura was left open, and a petrolatum (vaseline®) pack was placed extradurally as a drain. The wound was then closed tight except around the drain at the lower angle.

Postoperative Course.—The patient was given 300,000 units of penicillin twice daily and 1 gm. of sulfadiazine with sodium bicarbonate every four hours. Immediately after operation her temperature spiked to 104.2 F., but by the end of the first postoperative day it was down to 99.6 F., and after the third postoperative day it remained normal. On the eighth postoperative day the patient began to void. Thereafter, her general course was uneventful, and she was dismissed on the 11th postoperative day. Her convalescence at home was rapid and her recovery complete.

COMMENT

Incidence.—We have been able to find only six verified cases of acute subdural spinal abscess in the English literature. The present case is the seventh. Of the seven patients, five were males and two were females. The ages ranged from 14 to 52 years.

Pathologic Features.—The only offending organism isolated was the Staph. aureus. It was identified in five of the seven cases. The abscesses ranged in size from microscopic to abscesses containing 30 to 40 cc. of pus. The associated inflammatory tissue varied from a small amount of local reaction to involvement of the dura in its entire length. Most of these subdural abscesses were found in the lower thoracic and lumbar areas. One, however, occurred at the fourth thoracic vertebral level.

The abscess is usually considered to be secondary, although it may be difficult to establish the primary source of the infection. The primary focus may be forgotten by the patient, as it was in the present case, but it may be found if specifically sought for in the history. Furuncles, having been noted in four cases, might be considered

as the commonest precursor. Other possible sources of infection may have been lumbar puncture, pneumonia, osteomyelitis, and pyorrhea alveolaris. Chi [2b] noted three possible routes by which the offending organism can set up inflammatory reactions in the spinal meningeal spaces: (1) "metastasis through the blood stream," probably the commonest; (2) "direct extension of a contiguous infection in the neighborhood of the vertebral canal," including infection via contaminated lumbar puncture, and (3) "extension of infection along the sheaths of spinal nerve."

Intraspinal abscess produces significant changes in the spinal cord and adjacent nerve roots at the level of suppuration. It has been fairly well established [3] that deformity of the cord by compression does not occur to any significant extent. On the other hand, it has been demonstrated [4] that obstruction to the circulation of the cord is responsible for the changes in the cord, with resultant clinical manifestations.

Clinical Picture.—The clinical picture of acute subdural spinal abscess usually presents certain characteristic symptoms and signs (Table). A history of a preceding local infection (five cases) may be helpful when elicited. The illness often begins with localized back pain (five cases) and tenderness. There usually follows evidence of progressive involvement of the spinal cord or nerve roots, with sensory defects (six cases), impairment of motor function (six cases), sphincteric disturbances (seven cases), or any combination of these findings. In addition, subarachnoid block (six cases) is usually encountered. Fever, of varying degrees, is always present. It is to be noted that the clinical picture of subdural spinal abscess is similar in all respects to that of extradural spinal abscess. Differentiation of the two has been made only at the time of operation or at autopsy.

Lumbar puncture is an important aid in establishing the diagnosis of intraspinal suppuration. If the abscess is in the lumbar region, it may be punctured by the spinal needle. If the abscess is at another level, however, the cerebrospinal fluid may show only signs of an inflammatory reaction. Of the five cases in which the subdural abscess was not entered by the spinal needle, the cerebrospinal fluid showed an increase of leucocytes in two cases. The leucocyte count was 5 per cubic millimeter in one case and was not noted in two cases. Manometric studies revealed a subarachnoid block in cases in which the studies were done, and there was always an associated elevation of the cerebrospinal-fluid protein. The block was confirmed and located by myelography in the few cases in which this procedure was carried out.

The various types of myelitis, such as acute infectious myelitis and syphilitic myelitis, compose the main group of diseases from which an intraspinal abscess may have to be differentiated. The presence of subarachnoid block should exclude the inflammatory conditions of the cord, as well as meningitis or osteomyelitis of the vertebrae. Neoplasms of the cord, the meninges, or the vertebrae will not be accompanied by evidences of infection, which are always present in cases of intraspinal abscess.

Treatment and Prognosis.—When the diagnosis of intraspinal abscess is suspected, the only treatment is immediate laminectomy with drainage of the abscess. Needless to say, surgical measures should be supplemented with intensive antibiotic therapy.

3. Browder, J., and Meyers, R.: Pyogenic Infections of the Spinal Epidural Space: Consideration of Anatomic and Physiologic Pathology, Surgery **10**:296 (Aug.) 1941.

4. Suh, T. H., and Alexander, L.: Vascular System of the Human Spinal Cord, Arch. Neurol. & Psychiat. **41**:659 (April) 1939.

Summary of Cases of Subdural Spinal Abscess

Case No.	Author, Date	Incidence Sex Age	Pathologic Data Organism Source of Organism Site of Abscess, Approximate Description	Clinical Picture Symptoms and Signs*	Laboratory Data Lumbar Puncture Myelography	Treatment and Outcome Surgical Procedure Relation to Hospital Days	Treatment and Outcome Immediate and Eventual Relation of Death to Onset
1	Bennett and Keegan	M 32	Staph. aureus Furunculosis and/or osteomyelitis Subdural abscess, T 4 & 5 Small amount of pus and granulation tissue	Upper back pain for 5 wk.; difficulty in walking, paresthesia, sphincter disturbance, paraplegia, toxemia; sensory level from T 4; hyperreflexia	L. P.: complete block; w. b. c. ?; protein, 450 mg./100 cc. Myelogram: block at T 4	Laminectomy T 3, 4, 5 After several H. D.†	Dismissed with cane, 42d H. D. Essentially complete recovery
2	Arnett 1930	M 14	Staph. aureus Repeated lumbar punctures Subdural abscess, L 4 30 to 40 cc. of pus	Meningococcic meningitis for 5 days; daily lumbar punctures; generalized pain, leg pains; infection at first puncture site; cerebrospinal fluid thicker and thicker; sensory level from L 1, hyporeflexia, sphincter disturbance	L. P. for meningitis, increasingly thick cerebrospinal fluid, flow finally blocked by pus on 27th H. D.	Laminectomy L 3 28th H. D.	Dismissed with weak leg, 55th H. D. Complete recovery
3	Chi 1936	M 45	Staph. aureus Pyorrhea alveolaris and/or furunculosis Subdural abscess, T 10 3 cm. in diameter and granulation tissue	1st admission: iliosacral pain for 2 mo.; fever, numb thigh; neurologic examination and cerebrospinal fluid normal; remission of symptoms 2d admission (2 mo. later): hip pain for 6 wk.; weak and numb leg, hypesthesia, hyperreflexia, toxemia, sphincter disturbance, paraplegia, hyporeflexia	1st L. P.: normal 2d L. P.: block, 10th (?) H. D.; w. b. c. 5 per cu. mm.; proteins high Myelogram: block at T 10	Laminectomy T 7, 8, 9, 10 14th H. D. of 2d admission	No return of function Died 30th H. D. (2 wk. postoperative) 5 mo. after onset
4	Abbott 1940	M 51	Organism not cultured Source? Lumbar punctures? for syphilis Subdural abscess T 12 and L 1 Secondary leptomeningitis	Weakness and paresthesia of leg for 1 wk.; ascending numbness, hyporeflexia, sphincter disturbance, paraplegia Sensory level from L 1; leg pains Sensory level from T 8; bronchopneumonia and terminal meningitis	L. P.: "fluid dripped freely"; w. b. c. 15 and 33/cu. mm.; proteins ?; Wassermann reaction negative (blood Wassermann reaction positive)	No surgery (Treatment for syphilitic myelitis)	Died 41st H. D., 2½ mo. after onset
5	Freedman and Alpers 1948	M 36	Staph. aureus Furuncle and/or pneumonia Subdural abscess, T 9-L 1 Drops of pus; involvement of entire dura	Back pain and tenderness for 2 days; toxemia, severe back spasm, hyperesthesia of leg, abdominal and leg pain, sphincter disturbance; paresis, hyporeflexia; sensory level from T 10	L. P.: block, 10th H. D.; w. b. c. 4 and 1,600/cu. mm.; proteins, 2,960 mg./100 cc.	Laminectomy T 11, 12, L 1 22d H. D.	Died 23d H. D. (1st postoperative day) 3 wk. after onset
6		F 52	Culture sterile Pneumonia Subdural abscess, T 10-L 2 Microscopic size; granular mass; cord destruction, T 2 down	Intermittent back pain for 1 mo.; weak and numb legs, sphincter disturbance; toxemia, paraplegia, hyporeflexia; sensory level from T 4; condition too poor for operation, deterioration	L. P.: block, 5th H. D.; w. b. c. ?; proteins 1,200 mg./100 cc.	No surgery (Patient poor surgical risk by 21st H. D.; treated with penicillin)	Died 120th (?) H. D. 5 mo. after onset
7	Mufson and Solomon	F 30	Staph. aureus Furuncle Subdural abscess, L 1-S 2 Pockets of pus	Excruciating low-back pain for 9 days; sphincter disturbance, toxemia; tender and spastic back; neurologic status essentially normal	L. P.: block by pus 4th H. D.; no cerebrospinal fluid	Laminectomy L 2, 3, 4, 5; S 1 4th H. D.	Discharged 11th H. D. Complete recovery

* Approximately in chronological order from onset prior to hospitalization.
† H. D. indicates hospital day.

In addition to the present case, complete recovery was noted in two cases of subdural spinal abscess reported in the literature. These two cases [5] were the first and second reported in the literature, one in 1927 and the other in 1930. The patients did not have the benefit of present-day antibiotic therapy but survived, owing, undoubtedly, to the rapidity of surgical intervention. In the two cases of death following laminectomy and in one case in which the patient eventually became too ill to undergo operation, delay in surgical treatment was in large measure responsible for the fatal outcome. It is obvious, therefore, that the diagnosis of acute subdural spinal abscess must be considered early, for it is only after prompt surgical intervention that a clinical cure can be obtained.

SUMMARY AND CONCLUSIONS

1. A case of acute subdural spinal abscess is added to the six cases in the English literature, and a tabulated review of these cases is presented.

2. The clinical manifestations of acute subdural spinal abscess appear to be identical with those of acute extradural spinal abscess. They consist of back pain, followed by signs of a spinal-cord lesion with subarachnoid block and associated evidences of infection.

3. It is emphasized that prompt surgical drainage of the abscess is the only satisfactory treatment.

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5. Bennett and Keegan.[1] Arnett.[28]

LANGUAGE BEHAVIOR IN MANIC PATIENTS

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SPOKEN language is one major aspect of the total behavior pattern of a patient. Apart from the meaning of the words, one may obtain cues as to the patient's thoughts and emotions from the characteristics of speech itself. Exact methods of observation and analysis of speech may prove to be useful for better understanding of psychological reactions.

The patient uses words with regard to their meaning, relates words and sentences to each other, conveys some impression to the listener. These activities can be roughly paralleled with Carnap's [1] postulates of the functions of language as semantic, syntactic, and pragmatic. Where an obvious distortion in the use of language is apparent, as is so clearly manifest in the mentally ill patient, a descriptive localization of the defect within these areas should theoretically be possible. One of us (S. C.) [2] has outlined the possibilities of examining speech for psychological trends and diagnostic speech patterns and has stressed that relatively simple methods of language analysis have been little studied.

The aim of the present study is to determine how the analysis of the structure of speech may be made a useful preliminary to the study of function, and to explore the possibility of describing language behavior at this level in a group of patients in whom language distortion is prominent.

Language distortion is clinically evident in patients arbitrarily grouped into such general reaction types as schizophrenic, manic, and obsessional. It is not necessary to assume that language defect is correlated with diagnostic entities. Yet it is an observable fact that the language productions of patients in any one of these groups have more in common with each other than with those of the other groups or with "normals." To find wherein this difference lies, to what degree and in what manner it is shown, may provide an objective indicator of the kind of defect present in thinking, feeling, or attitude. Therefore it seems worth while to analyze the structure of language.

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TABLE 1.—*Individual 1,000-Word Samplings of "Manic" and "Normal" Speech, Analyzed for Distribution of Words as Parts of Speech* *

Individual 1,000-Word Samplings	Substantives		Adjective		Adverb		Main Verb		Auxiliary Verb		Pronoun		Prepositions		Conjunction		Article		Interjections	
	N	M	N	M	N	M	N	M	N	M	N	M	N	M	N	M	N	M	N	M
1	20.5	17.1	15.5	12.7	15.7	16.4	15.1	18.4	4.9	8.4	17.6	20.6	12.1	11.1	11.9	9.7	8.2	7.6	7.0	4.1
2	18.9	17.0	14.7	12.3	15.5	13.8	14.4	17.6	4.6	8.3	16.9	20.2	11.7	10.4	11.5	9.5	7.4	6.6	5.8	3.8
3	18.6	16.9	13.6	10.5	15.3	13.7	13.7	17.0	4.6	6.9	15.3	20.0	11.1	9.9	10.4	8.6	7.2	6.4	3.1	3.0
4	17.4	15.6	12.7	10.2	13.0	12.5	13.3	16.8	4.3	6.5	14.5	19.4	11.1	9.0	10.3	7.9	7.2	6.3	2.5	2.1
5	16.9	15.1	12.3	9.7	12.3	12.2	13.2	16.2	3.9	6.3	14.2	18.8	10.7	8.4	9.5	7.1	6.2	6.0	1.8	2.0
6	16.3	14.9	11.5	9.6	11.4	11.1	12.6	16.1	3.5	5.8	14.1	18.4	10.3	8.3	8.5	7.1	6.1	5.8	1.1	1.5
7	15.8	14.1	11.4	9.1	11.3	11.0	12.6	15.0	3.4	5.3	13.0	17.7	9.7	7.1	8.2	7.0	5.5	5.5	0.8	0.5
8	15.7	14.0	10.5	8.3	11.3	9.4	12.3	15.0	3.4	5.1	10.9	17.1	9.6	6.8	7.0	6.4	5.5	5.3	0.6	0.1
9	14.1	13.2	10.3	6.7	10.2	9.1	12.0	14.6	3.3	5.0	10.7	16.0	8.8	6.4	6.0	5.9	5.2	5.2	0.4	0.1
10	12.9	10.9	9.1	6.5	9.1	8.1	11.0	13.9	2.5	4.2	10.5	13.9	8.6	6.0	5.8	5.7	5.1	3.8	0.4	0

* Values arranged in order of decreasing magnitude and expressed as percentages.

METHOD AND OBSERVATIONS

For this study, 10 patients[3] clinically showing a manic type of behavior, with increased verbal productivity and pressure of speech, were selected. These patients maintained contact with their environment through the medium of intelligible speech, although the ideas and meanings behind their flow of talk were often obscure. In order that a basis for comparison might be introduced, samplings were also obtained from 10 subjects without mental illness and without any obvious language peculiarities. The second group consisted of persons of high-school and college level in education, as did the group of manic patients. Men and women were included in both groups. Beyond this similarity, no attempt to parallel the groups was made. The purpose was to obtain comparable samplings of speech from the two groups. The concept of a "control" group cannot be maintained when there are infinite variables. The second group serves to orient the investigator and to provide him a base line from which to observe. The technique of the interview situation was made as constant as possible, but variables, of course, occurred in that the manic patients talked quite spontaneously about the topics of their choice. Since in all instances their preoccupation centered to a large extent about their own life history and past experiences, the "normal" group was asked to talk within these areas. Samplings of 1,000 consecutive words were taken at random from a 15-minute recording of spontaneous flow of speech of each of the 20 subjects. These were transcribed and analyzed.

TABLE 2.—*Word Frequency Distribution in "Normal" and "Manic" Speech Based on Ten 1,000-Word Samplings*

	Normal Group	Manic Group	P *
Ten Most Frequently Used Words	I, and, the, to, a, of, in, that, my, was	I, and, the, to, a, of, in, that, it, was	....
No. of different words having rank-frequency distribution of 1-10	31	42	....
Recurrence of most frequently used word ("I") per 1,000 words	66	69	0.05
No. of words used once per 1,000 words	200	183	0.05
No. of different words per 1,000 words	341	315	0.05-0.02 †

* P denotes probability value, as determined by the t test.
† means statistically significant.

In terms of grammatical construction, uniformity in analysis was maintained by having the same person qualify the words as parts of speech. The t test, as an application of the critical ratio to small samples, was applied wherever feasible, and the data which have statistical significance on the basis of this test are starred (Tables 2 and 3). This test takes into account the increasing unsatisfactoriness of representative measurements as the size of the sample decreases.

Table 1 represents individual 1000-word samplings of "manic" and "normal" speech, analyzed for distribution of words as parts of speech and arranged in order of decreasing magnitude. The use of words as parts of speech seems to fall within a fairly uniform distribution range for the samples of each series. When a series of "manic"-speech samplings is compared with a series of "normal"-speech samplings, it is noted that there is considerable overlap with the exception of auxiliary verbs and pronouns. In the use of auxiliary verbs only one sampling of "manic" speech was lower than the highest of the "normal" group; in the use of pronouns only three samplings of "manic" speech were lower than the highest of the "normal" group.

Table 2 is based on calculations of the rank-frequency distribution of words. Determinations were made by counting the occurrence of each different word in

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each 1,000-word sampling, assigning to the word used with greatest frequency the rank 1, the next most frequently used word, rank 2, and so on. For example, in one sampling of manic speech, "I" was used 82 times and was given rank 1; the word used next most frequently, "the," occurred 55 times and was given rank 2. Table 2 represents a summary of the significant findings.

The 10 most frequently used words are identical for the two groups with one exception. More different words having a rank-frequency distribution of 1 to 10

TABLE 3.—*Recurrence of Words as Parts of Speech in Ten 1,000-Word Samplings of "Normal" and "Manic" Speech, Expressed in Percentages*

	Normal	Manic	*P* *
Substantives	16.7	14.9	0.1-0.05
Adjectives	12.2	9.6	0.02-0.01 †
Adverbs	12.5	11.7	0.5-0.4
Main verbs	13.0	16.1	>0.01 †
Auxiliary verbs	3.8	6.2	>0.01 †
Pronouns	13.8	18.2	>0.01 †
Prepositions	10.4	8.3	>0.01 †
Conjunctions	8.9	7.5	0.2-0.1
Articles	6.4	5.9	0.3-0.2
Interjections	2.4	1.7	0.5-0.4
Conceptual/articulatory ratio	1.19	1.09	0.3-0.2
Verb/adjective quotient	1.07	1.68	>0.01 †

* *P* denotes probability value, as determined by the *t* test.
† Means statistically significant.

TABLE 4.—*Temporal Pattern of Speech as Determined Through Verzeano Speech Analyzer**

		Mean	Range	*F* *	Significance
Number of units per minute	Controls	17.9	15.9-20.9	6.719	Significant at
	Manics	19.5	10.5-24.3	.....	1% level
Duration of units of speech	Controls	2.148	1.618-2.643	7.919	Significant at
	Manics	1.985	1.128-4.939	.....	1% level
Duration of units of silence	Controls	1.195	0.957-1.405	10.111	Significant at
	Manics	1.321	0.838-2.360	.....	1% level
Number of words per minute	Controls	154.3	127.8-189.5	4.644	Significant at
	Manics	151.4	88.8-216.2	.....	5% level

* Analysis of variance, using the *F* test, was significant in every case.

are utilized in "manic" speech than in "normal" speech. The recurrence of the most frequently used word, "I," is slightly, but not significantly, higher in "manic" speech. The number of words used once per 1,000 words is slightly, but not significantly, lower in "manic" speech. The number of different words used per 1,000 is significantly lower.

Table 3 represents a comparison of words as parts of speech in the two groups. In "manic" speech significantly greater emphasis is given to the use of verbs, particularly auxiliary verbs, and pronouns. There is a decrease in the use of adjectives and prepositions. Despite the shift in emphasis, the balance of articulatory and conceptual words remains roughly the same in the two groups. There is a significant increase in the verb-adjective quotient in "manic" speech.

In a study of the physical characteristics of speech [4] in these subjects, through the Verzeano Speech Analyzer,[5] from which the temporal pattern of speech can be obtained, the words per minute, units of speech, and units of silence showed significant differences in the two groups (Table 4). The difference lies in the range, or spread, of variation. The rate of words per minute varied from 88.8 to 216.2 for the "manic" group and from 127.8 to 189.5 for the "normal" group. The remarkably low rate for one of the manic patients is explained by the fact that this patient, although showing the usual inclination to speak with little or no stimulus and a continuous flow of words, used a deliberate, slow declamatory style. This patient's speech is representative of a type of verbal behavior sometimes seen in manic patients. The units of speech and units of silence per minute (0.5 second being used as an arbitrarily selected pause between units) also reflected a greater range of variation in "manic" speech. These findings reinforce each other in pointing to a variable, inconstant speech pattern, in contrast to a more predictable and uniform pattern in "normal" speech. A study of speech as a form of language behavior is now in progress and will be amplified and discussed in a subsequent publication.

REVIEW OF LITERATURE

Within the last half-century some attempts have been made to integrate the broader concepts of language into the field of neuropsychiatry. In his classic work relating language to the thought processes of the child, Piaget [6] demonstrated an inherent structure or pattern in the development of speech in which there is progressive differentiation from egocentric to socialized speech. Stinchfield's [7] monograph on speech disorders takes cognizance of the area of speech pathology that is correlated with psychological determinants and indicates that speech disorders are frequently one of the first diagnostic signs of mental disorders. Goldstein's survey [8] (Language and Language Disturbances), although oriented toward the psychology of aphasia, presents his theories regarding language function in general. He postulates language as consisting of two types, speech automatisms and abstract language, and indicates that disorders involving so-called higher mental processes and emotion could be investigated by studying deflections in language under these two aspects.

Zipf [9] presents a somewhat similar view by differentiating between articulatory and conceptual words. Words as parts of speech fall into two distinct groups. In Group 1 (conceptual) are the substantives, adjectives, adverbs, and main verbs—a group containing many different words and having a low ratio of recurrence. In Group 2 (articulatory) are auxiliary verbs, pronouns, prepositions, conjunctions —parts of speech containing but a few different words and having a high ratio of

4. Technical assistance of Mr. Paul Johnson.

5. Verzeano, M., and Finesinger, J. E.: An Automatic Analyzer for the Study of Speech in Interaction and in Free Association, Science **110**:45-46, 1949. Verzeano, M.: Time Patterns of Speech in Normal Subjects, J. Speech & Hearing Disorders **15**:197-201, 1950.

6. Piaget, J.: The Language and Thought of the Child, New York, Harcourt, Brace and Company, Inc., 1932.

7. Stinchfield, S. M.: Speech Disorders: A Psychological Study of the Various Defects in Speech, New York, Harcourt, Brace and Company, Inc., 1933.

8. Goldstein, K.: Language and Language Disturbances, New York, Grune & Stratton, Inc., 1948.

9. Zipf, G. K.: The Psycho-Biology of Language, Boston, Houghton Mifflin Company, 1935.

recurrence. The author recognizes speech as a part of behavior which can be studied objectively. He points to two possibilities by which language may be studied—by frequency of word use and by study of the elements of speech—and he makes the distinction between the kinds of words used, whether reporting data of experience or relating these data to each other. In normal speech the sort of equilibrium is set up which does not go too far in either direction but keeps communication understandable. He calls attention to the fact that in pathological speech this equilibrium is upset in some way. Later Whitehorn and Zipf [10] demonstrated, in a study of three mentally ill patients, that the balance between articulatory and conceptual words may be correlated with tendencies toward repetitiveness and diversification, and that this holds, in turn, with the egocentric and the allocentric orientation of the subject.

More recent investigation of the use of language by psychotic and neurotic patients falls broadly into three categories: (1) descriptive enumeration of peculiarities of speech; (2) discussion of semantic values, and (3) attempts to correlate language pattern with specific emotional states. The first group is exemplified by the description of speech found in textbooks of psychiatry. A more critical analysis is that of Newman and Mather,[11] who have studied the spoken language of patients with affective disorders from the standpoint of the physical qualities of speech, as well as vocabulary, style, and syntax. Study of the meaning of words and the use of word symbols, mainly in schizophrenic patients, is well documented but does not fall within the scope of this paper. Some investigators have focused on the possible correlation between the use of certain speech elements and emotional states or personality traits. Ruesch and Prestwood [12] have noted an increase in the use of personal pronouns and subjective qualifications in states of emotion and self-concern. In a second paper,[13] these authors indicate that psychosomatic illnesses have in common features which express lack of maturation and retardation of the development of higher processes of communication. Balken and Masserman [14] have found a high verb-adjective quotient when the anxiety state is the highest. Mayers and Mayers [15] note a lack of articles and prepositions as a precursor to loss of spatial orientation and describe the passive and depressed patient as an omitter of verbs and adjectives. Feldman [16] points to the use of locutions as a crutch to overcome difficulty arising from anxiety.

Dynamic philology and linguistics [17] demonstrate that language behavior can be described in terms of orderly pattern and structure constant to language as such,

10. Whitehorn, J. C., and Zipf, G. K.: Schizophrenic Language, Arch. Neurol. & Psychiat. **49**:831 (June) 1943.

11. Newman, S., and Mather, V. G.: Analysis of Spoken Language of Patients with Affective Disorders, Am. J. Psychiat. **94**:912, 1938.

12. Ruesch, J., and Prestwood, A. R.: Anxiety: Its Initiation, Communication and Interpersonal Management, Arch. Neurol. & Psychiat. **62**:527 (Nov.) 1949.

13. Ruesch, J., and Prestwood, A. R.: Communication and Bodily Disease: A Study of Vasospastic Conditions, A. Res. Nerv. & Ment. Dis., Proc. (1949) **29**:211-230, 1950.

14. Balken, E. R., and Masserman, J.: The Language of Phantasy, J. Psychol. **10**:75, 1940.

15. Mayers, A. N., and Mayers, E. B.: Grammar Rhetoric Indicator, J. Nerv. & Ment. Dis. **104**:604, 1946.

16. Feldman, S. S.: Mannerisms of Speech, Psychiat. Quart. **17**:356, 1948.

17. Bloomfield, L.: Language, New York, Henry Holt & Company, Inc., 1941. Jesperssen, O.: Language: Its Nature, Development and Origin, New York, Henry Holt & Company, Inc., 1922. Sapir, E.: Language, New York, Harcourt, Brace and Company, Inc., 1921.

and to a degree independent of individual variants. There is evidence of orderliness and consistency in the internal structure of language in terms of balance and distribution of words.[9] Curves for word-frequency distribution in the English language show a consistent pattern.[18] Frequency of word usage, the recurrence of the various elements of speech is, although flexible, not random.

COMMENT

The findings here reported support the thesis of Zipf and others that a given language shows an inherent, although flexible, consistency in its pattern which can be demonstrated despite the variables introduced by different speakers. The fairly consistent distribution scatter of words as parts of speech in the two groups would seem to indicate that an automatic and roughly predictable pattern of word usage underlies the "manic," as well as the "normal," speech production, although some variations do occur in the former. This observation is reinforced by the demonstration that the 10 most frequently used words are practically identical and that the balance between conceptual and articulatory words remain about the same. In the process by which language becomes the motor expression of thoughts, the speaker unconsciously adheres to certain laws of word distribution and balance. In the group of manic patients studied, this automatic pattern, although showing some variation, was not disorganized to a marked degree, despite the fact that, on the basis of clinical observation, all these patients failed to convey fully intelligible ideas. The main distortion of "manic" speech would not seem to occur at the level of morphology.

The manic patient uses significantly fewer different words in discourse, and a greater number of the different words used fall within the group of words having a rank-frequency distribution of 1 to 10. The inference drawn from this observation is that "manic" speech shows a greater tendency toward homogeneity and repetitiveness than does "normal" speech. These findings may confirm Noyes's[19] observations that although superficially manic speech gives the impression of a great variety of ideas, actually the range is limited.

Perhaps the most striking phenomenon that appears in these findings is the relative increase in the use of pronouns, main verbs, and auxiliary verbs and the relative decrease in the use of adjectives and prepositions.

In the stream of speech pronouns are ordinarily increased when there is recurrence of a given topic. This would be consistent with the inference drawn above. Personal pronouns, the predominance of "I," would seem to point to a self-orientation or preoccupation of the speaker. The substitution of a pronoun for a name would seem to make the boundaries of an object more vague, to differentiate less clearly the limitations and specificity of the object or idea. The object would have less independent meaning in its own right but would accumulate the implied meanings and values of the speaker. A corresponding decrease in the use of adjectives would also appear to reflect a lessened degree of awareness of, or emphasis upon,

18. Zipf, G. K.: Relative Frequency, Abbreviation and Semantic Change: Selected Studies of the Principle of Relative Frequency in Language, Cambridge, Mass., Harvard University Press, 1932.

19. Noyes, A. P.: Modern Clinical Psychiatry, Philadelphia, Ed. 3, W. B. Saunders Company, 1948.

the qualifying or differentiating characteristics of an object or idea. Possibly these findings may lend support to the impression that manic patients are less concerned with communication than is the average person in normal discourse.

Verbs suggest action or relate the data of experience to each other or to the speaker. Coupling with auxiliary verbs (was, will, has, can, etc.) introduces a time element (reference to past, future, probability) or change in voice (active or passive). An increase in verbs is obviously complex in meaning, but suggests that a detailed study of this phenomenon may be an index to the attitude or point of view of the individual patient. Our findings in respect to an increased verb-adjective quotient in manic patients parallel those of Balken and Masserman for patients with a high degree of anxiety.

It is not quantitative distribution alone which determines the character of the speech pattern. The intensity and emotional value of a word used lend richness and individuality to speech. This is a flavor with which some manic patients season their language. It may make up for the smaller number of qualifying words used. In no written report can the important elements of voice be reproduced. Pitch, modulation, rhythm, etc., can be studied, but require other techniques. They are certainly of great importance, especially in evaluating emotional variations.

SUMMARY AND CONCLUSIONS

Specific findings in analyzing the speech of 10 manic patients were as follows:

1. There was persistence of the general framework inherent in spoken language. This was shown in terms of (*a*) the 10 most commonly used words; (*b*) the distribution range of various speech elements, and (*c*) the balance between conceptual and articulatory words.

2. There was a tendency to use fewer different words and to repeat these different words with greater frequency than in "normal" discourse.

3. There was a quantitative change in the use of certain speech elements, namely, (*a*) a relative increase in the use of pronouns, main verbs, and auxiliary verbs; (*b*) a relative decrease in use of adjectives and prepositions; (*c*) a high verb-adjective quotient.

4. The flow of speech in terms of rate, units of speech, and units of silence, was more uneven and was subject to wider variation than was "normal" speech.

On the basis of these findings, a tentative descriptive analysis of language behavior in manic patients is formulated as follows:

The language pattern of these manic patients indicates that gross disorganization does not appear at the level of structural elements and that the dynamic laws demonstrable for any given language are operating. It is postulated that the defect of manic speech occurs at higher integrative levels of language formulation. A shift in emphasis in the use of elements of speech occurs in the direction of repetitiveness and homogeneity and away from that part of the vocabulary which qualifies, differentiates, and individualizes. The interaction with the listener appears to be more characteristic of expressive behavior than of communication. If the assumption of a correlation between emotional states and verb-adjective quotient is correct, the manic patient's speech gives objective evidence of a heightened degree of anxiety. Greater variation, with less predictability and uniformity, occurs in the physical pattern of speech.

INTRACRANIAL ANEURYSMS

Clinicopathologic Considerations of Oculomotor-Nerve Regeneration and Intracerebral Hemorrhage

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THE LITERATURE concerned with saccular aneurysms of the cerebral arteries deals predominantly with the syndromes these lesions present, diagnostic procedures, of which arteriography receives a warranted emphasis, and a growing contingent of reports on prognosis and the results of surgical intervention. Pathologic studies concern chiefly the histologic processes responsible for aneurysmal formation, with a smaller number of reports on changes in the meninges and the topographic distribution of the hemorrhagic foci resulting from intracerebral hemorrhage. There is a dearth of information on the late changes in the nervous system following the various manifestations of cerebral aneurysm. It has been my good fortune to observe two patients with such residual lesions. In the first patient the oculomotor regenerative syndrome of the "pseudo-Graefe phenomenon," of comparatively insidious origin, was present, and the rootlets of the third cranial nerve showed definite evidence of axonal neoformation; in the other, a subcortical hemorrhagic cyst was disclosed.

OCULOMOTOR-NERVE REGENERATION

CASE 1.—Mrs. J. P. was first seen in 1946, at the age of 67, when she was referred by Dr. L. E. Allday because of episodes of confusion. She gave a history of recurrent, severe sick headaches since childhood, usually occurring unilaterally on either side of the head and unaccompanied with visual disturbance. In 1935 she complained abruptly of severe pain in the vertex and fell unconscious. The cerebrospinal fluid was bloody. After 18 hours she regained consciousness, and later made a seemingly complete recovery. A second attack of subarachnoid hemorrhage occurred in 1939, with sudden loss of consciousness, which lasted about 1½ days. When she regained consciousness, she had loss of central vision in the left eye; at the same time the family noted that the left upper eyelid drooped about halfway. However, the ptosis was never complete, and she did not complain of diplopia. The ptosis would recur at times, being of an equal degree to that at the onset. Since the second episode she had been noted to be less alert than before; her memory for recent events was impaired, and she became irritable, in contrast to her previously sweet and even disposition. Since 1941 she had had about six episodes of confusion, the last three within the three months preceding consultation. In these attacks she would wonder where she and her children were, and she stated that everything seemed to be in a dream. These attacks would last two or three hours.

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

On examination her blood pressure was 186/102. She was fairly alert and oriented. The left optic disk showed temporal pallor, and a large absolute central scotoma was noted in the left visual field. The pupils were equal, measured 2.5 mm. in diameter, and reacted moderately to light. The only defect noted in the ocular movements at that time was poor convergence. The grip of the right hand was slightly weak. The deep reflexes were moderately exaggerated in all extremities, with a bilateral Hoffmann sign, but the plantar responses were flexor. An electroencephalogram (taken when she was awake) showed a focus of 2- to 4-per-second waves in the left temporal and parietal regions. Roentgenograms of the skull revealed nothing abnormal. On the supposition that the attacks of confusion represented temporal-lobe discharges, she was placed under a regimen of small doses of phenobarbital. Four months later she stated that she had had only one attack; this had lasted a few seconds, during which time she was slightly disoriented.

She was next seen in 1947, an hour after she had been struck by a car, sustaining a fracture of the right femur, but with no evidence of head injury. At this time anomalous movements of the left eye were observed. The pupil of the left eye was slightly larger (2 mm.) than that of the right eye (1.5 mm.), both reacting fairly well to light. Whereas all movements of the right eye were full, only external rotation of the left eye was normal. Adduction of the left eye was weak and was accompanied with downward rotation; at the same time, the upper lid was raised appreciably, but there was no change in the pupil. Mild ptosis occurred on abduction. Slight upward and downward movements were present; the upper lid on the left side did not follow the globe on downward rotation as well as the lid on the right side. These changes were interpreted as a pseudo-Graefe phenomenon, although they differed from the usual picture of this condition in the pupillary reaction to light and the presence of vertical eye movements (Bielschowsky [1]).

Ptosis of the left upper eyelid continued to appear at various times, being apparently more pronounced when she was tired. At no time did she complain of unilateral frontal or ocular pain. She continued phenobarbital medication and had no further attacks of mental confusion. A third hemorrhage occurred in 1950, when she suddenly complained of excruciating headache, vomited, and soon became unconscious. The pupils were large and fixed. The cerebrospinal fluid was bloody. Her temperature rose to 105 F., and she died seven hours after the onset of the headache.

At autopsy (by Dr. J. L. Smith), a thick layer of blood was observed to cover the ventral surface of the brain stem, cerebellum, and frontal region. A large aneurysm (2.4 by 1 by 1 cm.) arose from the left internal carotid artery at the upper border of the junction with the posterior communicating artery and was directed posteriorly and slightly laterally, so that it overlay the posterior communicating and posterior cerebral arteries (Fig. 1). A subcortical hemorrhage in the left temporal lobe had ruptured into the inferior horn of the lateral ventricle, and the left ventricle was filled with blood, the other parts of the ventricular system containing a smaller amount of bloody fluid. In the center of the pontine tegmentum there was a minute hemorrhage. After fixation of the brain in dilute formalin, the left oculomotor nerve could not be identified beyond its roots in the interpeduncular space, frayed strands passing forward into the extensive blood clot surrounding the aneurysm. The right oculomotor nerve appeared normal, as did the other cranial nerves, and the optic chiasm was not displaced or distorted.

Microscopic study was made after the formalin-fixed tissue was sectioned in paraffin and stained with hematoxylin and eosin, and by Bodian's method for axis cylinders, Perdrau's method for reticulin,[2] Weil's method for myelin, Masson's trichrome method (Lillie variant, with fast green FCF), Mallory's phosphotungstic acid-hematoxylin, and Van Gieson's connective-tissue stain. Conspicuous lesions were observed in the left oculomotor nerve, the rootlets of which contained numerous cellular nodules in their peripheral segments. These were composed of spindle-shaped cells, arranged in parallel fashion in interlacing bundles, with a definite tendency for the nuclei to be arranged in rows or palisades (Fig. 2A). The nodules were elongated and discretely outlined. They often composed only a portion of the root fascicle, with thick

1. Bielschowsky, A.: Lectures on Motor Anomalies of the Eyes: II. Paralysis of Individual Eye Muscles, Arch. Ophth. **13**:33 (Jan.) 1935.

2. Miss Beatrice Kahn, of Dr. Percival Bailey's laboratory, made the Bodian and Perdrau preparations.

medullated fibers passing down the margin of the nerve, or in places penetrating the center of the cellular lesions. Rootlets containing these convoluted lesions were somewhat wider than the normal nerve bundles. The tissue was contained within the epineurium, the restraining influence of which seemed responsible for the complex interlacing pattern of the cell bundles.

Where the cells were oriented in the plane of section, their nuclei were uniformly plump, sausage-shaped, and regular in contour. They were vesicular and had a delicate membrane,

Fig. 1 (Case 1).—Basal cerebral arteries, with large saccular aneurysm of left internal carotid artery, arising at junction with posterior communicating artery (cut during the removal of the brain).

beneath which numerous fine chromatin granules were arranged; thus, when the nuclei were bisected, their centers appeared hollow. Nucleoli were inconspicuous. The nuclei closely resembled the Schwannian nuclei of normal nerve bundles, except that the latter were often rounded and their nucleoli somewhat more prominent. Details of these cells were seen in preparations stained by Bodian's method, in which they were faintly colored a reddish brown. They formed very long, slender protoplasmic structures (Fig. 3*B*). These were rather straight except for

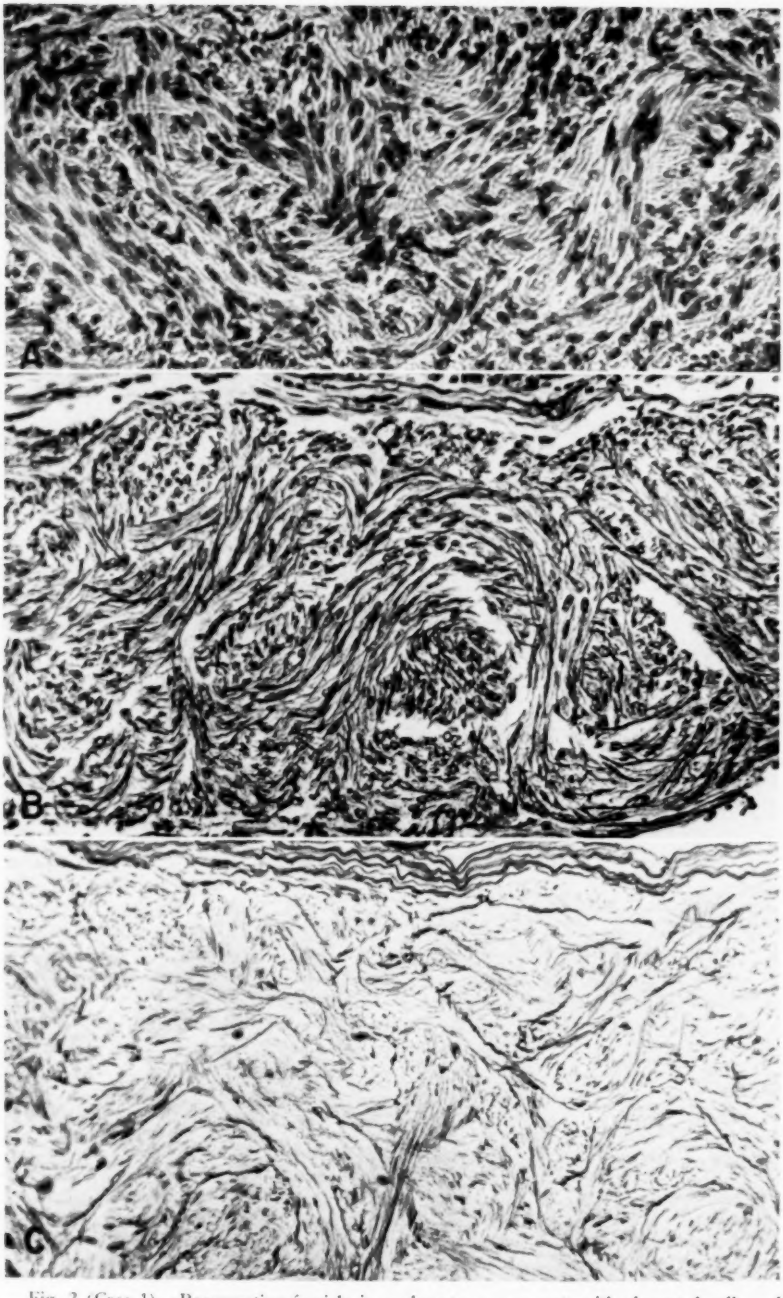

Fig. 2 (Case 1).—Regenerating fascicle in oculomotor nerve root, with elongated cells and fibers arranged in interlacing bundles.

A, appearance with routine cell stain, showing palisading of nuclei; hematoxylin-eosin stain; ×220.

B, nerve-fiber preparation, showing the prominent fiber component. At top of figure is a bundle of irregular, but otherwise intact, nerve fibers. Bodian's method; × 220.

C, reticulum preparation. Reticulin fibers are fewer and more delicate than the axons. At the top the endoneurium is densely impregnated. Perdrau's modification of Bielschowsky's method; × 220.

Fig. 3 (Case 1).—*A*, spiral formation of neurites and cells, arranged about minute hollow tube. Above is portion of retiform tissue (fasciculated tissue in cross section), composed of tubes, fibers, and nuclei. Bodian preparation, counterstained with Masson's trichrome stain.

B, elongated cells (tubes) incorporating nerve fibers. Note varicosities on the fibers. Bodian's stain; × 1,500.

tapering processes, which frequently were tortuous. Branching was at times so profuse as to resemble a partially opened fan. The cells were somewhat disarranged where they were loosely assembled, but approached a parallel disposition when more closely packed. They did not form whorls, except for a single spiral formation of neurites lying within concentric layers of collagen fibers, between which were scattered oval nuclei and delicate cytoplasmic filaments, all surrounding a narrow central tubule (Fig. 3*A*). With Masson's stain, the cell bodies resembled hollow tubes of varying thickness, with a delicate ponceau-fuchsin-staining cell membrane (Fig. 4). Other types of cells were less frequently noted. The nuclei of some were rod-like, densely

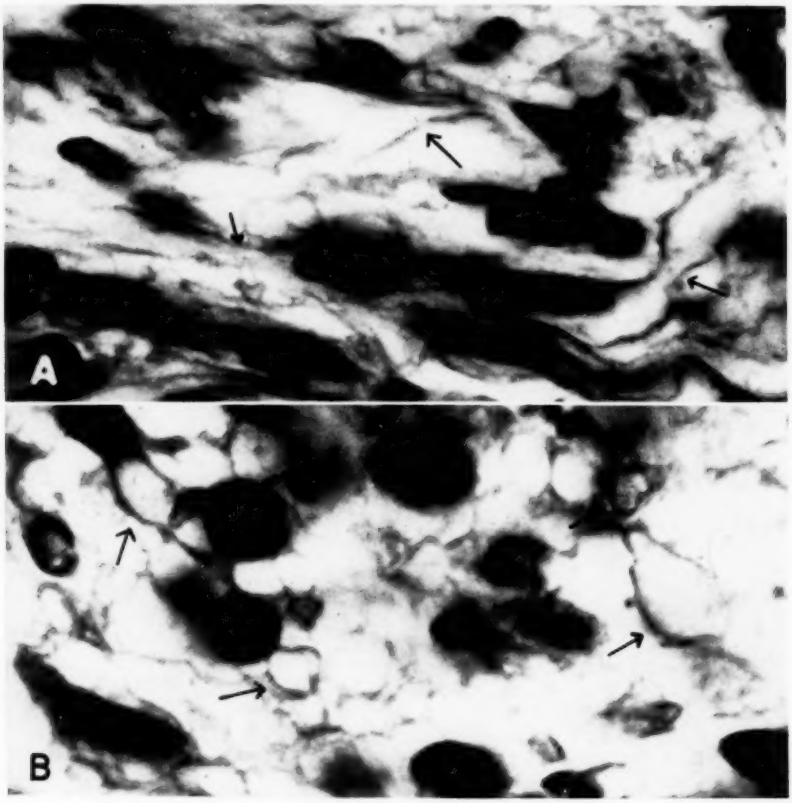

Fig. 4 (Case 1).—Cytoplasmic tubes, several of which are indicated by arrows. *A*, tubes seen in longitudinal section, one passing across lower center, bending upward at the right side. *B*, transverse section of fenestrated collagenous tissue, only some of the spaces being filled by the cytoplasmic tubes. Masson trichrome stain; × 1,700.

staining, and either straight or crinkled; in other cells they were fairly rounded and dense and were surrounded by a clear halo. There were no definite phagocytes. Mitotic figures were not seen.

In a considerable portion of the nodules the fasciculated tissue was sectioned transversely, appearing as fenestrated tissue with rounded nuclei, from which delicate collagenous strands radiated, to mingle in retiform fashion with one another (Figs. 4*B* and 5). The spaces varied considerably in size, the largest having the width of the nucleus. The nuclei were situated within

these canals; in many instances they were partially surrounded by a clear space, although in some canals no such perinuclear space was seen. Cytoplasmic membranes lined only some of the canals; others appeared merely as empty spaces within the collagenous network.

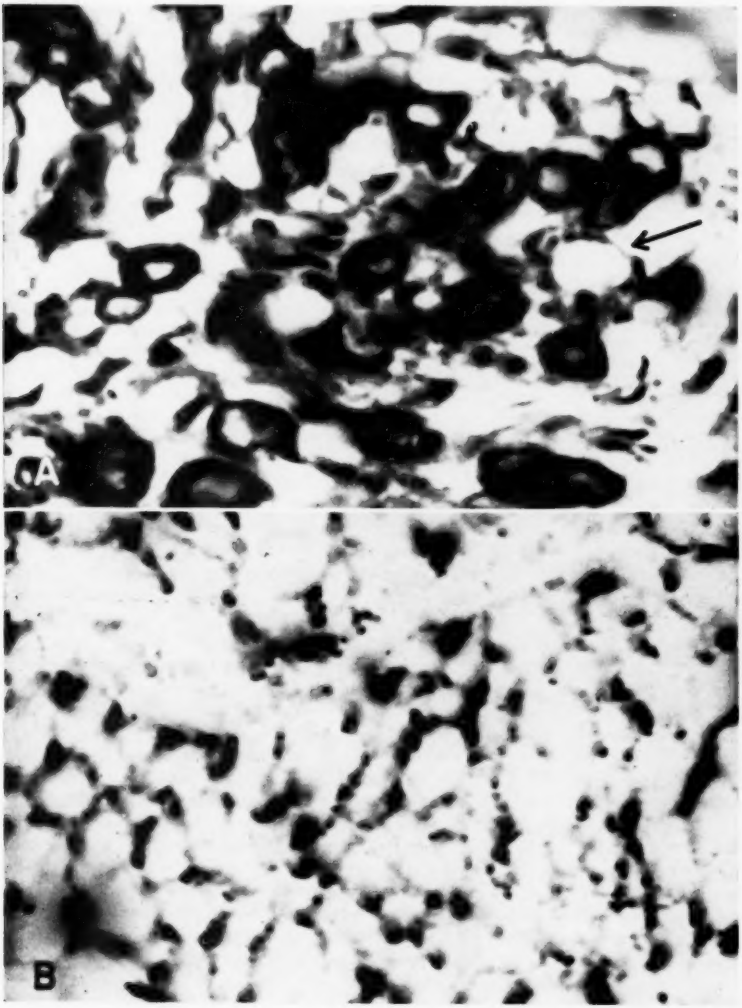

Fig. 5 (Case 1).—Relations of fibers to fenestrated tissue, as seen in transverse section. *A*, neurites embedded in the lining of tubes, projecting slightly into the lumen. This is best seen in the tube at the right (indicated by arrow). Bodian's method with Masson's counterstain; × 1,700.

B, reticulin fibers passing within the collagenous sheaths. Perdrau's stain; × 1,700.

A profusion of fibers was demonstrated by impregnation methods. The fibers were arranged in interlacing bundles similar to those noted in cell preparations, parallel to the long axis of the

cells. The use of the Bodian and Perdrau techniques indicated that two types of fibers were present. In both, the fibers were of variable caliber. Those revealed in Bodian preparations (Fig. 2*B*) were more numerous and generally thicker and displayed the fusiform varicosities of axons. Most of these fibers were rather straight, but some were wavy, or even spiral (Fig. 3*A*). A few of the fibers gave rise to fine sprouts. Stained black, they were usually seen against a background of the elongated cells, to which they adhered closely (Fig. 3*B*). However, the association of axons and cells was not constant; while some cells had two or three fibers, others had none. Myelin was not seen within the cellular lesions; medullated fibers existed only where the nodules shared a rootlet with normal longitudinal nerve fibers.

As stated, the reticulin composition (in Perdrau preparations) was less abundant (Fig. 2*C*) than the axis cylinders. The connective-tissue fibers were also considerably more delicate and quite regular in thickness over long distances. For the most part, they were tortuous or serpentine, in contrast to the nerve fibers, which were mostly straight. Connective-tissue stains, such as Masson's trichrome stain, revealed a considerably greater amount of collagen in delicate sheets and strands throughout the cellular tissue, which were more or less oriented with the other fiber and cell constituents.

The relations of cell, neurite, reticulin, and collagen were best understood by study of the reticulated portions of the nodules. The delicate strands comprising the network in these areas were unstained by either the Perdrau or the Bodian method, but gave a definite reaction for collagen with the various connective-tissue stains. Tiny round or oval granules were included within most of these partitions, representing cross sections of the respective fibers; here and there some would take a transverse course for a short distance, but they always followed the strands of the matrix. They were colored black in the metallic impregnations (Fig. 5) and dark red with Masson's trichrome stain, but were not revealed with the other stains. When Bodian preparations were counterstained with Masson's trichrome stain, many of the green collagenous structures were seen to be lined by a very thin protoplasmic membrane, within which were embedded the fiber particles (Fig. 5*A*). However, in other spaces of similar size, shape, and position, no such lining could be discerned, even though fibers were present. The particles of reticulin (in the Perdrau preparations) were minute, often appearing pinpoint in size, even under the highest magnification. In contrast to the nerve fibers, they were embedded within the collagenous strands, which was evident when the latter either were left unstained or were counterstained by Van Gieson's method (Fig. 5*B*). As was the case with the neurites, reticulin fibrils could not be demonstrated in some of the collagenous tubes.

The more normal roots showed mild changes in the peripheral segments. Some of the nerve fibers were irregularly swollen, and the Schwann nuclei were moderately increased. The longitudinal fibers of Key and Retzius were heavily impregnated by Perdrau's method, but the network of Plenk and Laidlaw was only partially revealed. Masson's stain gave the usual diffuse coloration of the endoneurial sheaths, which probably were slightly thickened.

The central, or glial, segment of the left oculomotor nerve also presented definite changes in its distal portion. There were large vacuoles containing loose debris. In these areas the axis cylinders were often bulbous, and a few fine Y-shaped fibers suggested that they had divided. Myelin sheaths had for the most part disappeared, stopping abruptly at the margin of the vacuolated portion, with only a few fine and irregular medium medullated fibers persisting here. Astrocytes were increased in number, and a fairly dense isomorphic glial fibrosis was present at the margins of the roots. The intramedullary portions of the nerve appeared normal, as did the cells in the caudal portion of the oculomotor nucleus, which was included in the sections. An incidental finding was a single large ganglion cell of encapsulated variety (Fig. 6) in the peripheral segment of a rootlet, included in a bundle of normal nerve fibers adjacent to one of the nodular lesions. This cell may be designated as an abortive heterotopic ganglion.[3]

The left temporal lobe adjacent to the aneurysm showed considerable degeneration. The cerebral cortex here appeared to have been destroyed, so that the subcortical white matter was denuded. The subcortex was rarefied and gliosed to a variable degree. In places the cytoplasm of

3. According to I. M. Tarlov (Structure of the Nerve Root: Nature of the Junction Between Central and Peripheral Nervous System, Arch. Neurol. & Psychiat. **37**:555 [March] 1937), this is not an unusual occurrence, even in motor nerves.

astrocytes contained fine particles of hemosiderin, but without phagocytes of the histiocytic series being in evidence. In some areas the cerebral cortex showed only a loss of neuronal elements, but in the walls of the collateral fissure the middle layers of the cortex had undergone gliosis and spongy disintegration, with a row of large, uniformly rounded phagocytes, heavily loaded with golden-brown pigment.

The left optic nerve appeared somewhat paler than the right in myelin-sheath preparations. The pallor was most pronounced in the center, where the fibers appeared reduced in number, although there was no discrete area of demyelination. Portions of the perineurial sheath of this nerve were infiltrated with fresh blood. Other cranial nerves (the right 3d, 4th, 7th, 8th, 9th, and 10th) showed no change in either the peripheral or the central segments. In the cerebellum there was mild perivascular infiltration with lymphocytes; cells of this type were present also in the subarachnoid space, buried between the folia, with an occasional binucleated form. The wall of the aneurysm was composed of the usual collagenous tissue. Additional pathologic changes were arteriosclerosis of the basal cerebral and coronary vessels, hypertrophy and fibrosis of the myocardium, pulmonary edema, chronic interstitial pneumonitis, and chronic hepatitis.

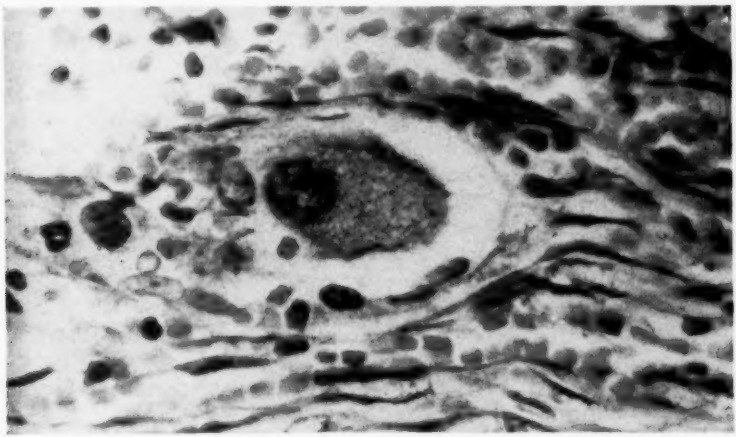

Fig. 6 (Case 1).—Encapsulated large ganglion cell within peripheral segment of a preserved oculomotor nerve rootlet, surrounded by axons, mostly thin. Bodian's method; × 700.

Comment.—Clinical Considerations: Clinically, the most interesting feature of this case was the development of the pseudo-Graefe sign without a clear oculomotor nerve paralysis ever having occurred. This phenomenon is now generally considered as a mass action resulting from an exuberant regeneration of nerve fibers following an acute lesion of the oculomotor nerve, the newly formed fibers growing indiscriminately to the various muscles supplied by the nerve (Ford, Walsh, and King [4]). One may presume that the injury in this case was of low intensity and recurrent, with only a small number of fibers involved at any particular time, too small to present gross loss of function.

4. Ford, F. R.; Walsh, F. B., and King, A.: Clinical Observations on the Pupillary Phenomena Resulting from Regeneration of the Third Nerve, with Especial Reference to the Argyll Robertson Pupil, Bull. Johns Hopkins Hosp. **68**:309, 1941.

The incompleteness of the nerve lesion in this case is probably the reason for the fragmentary character of the pseudo-Graefe complex of muscle action. This eponymic designation refers to the conspicuous lid lag and has no relation to other less obvious, though equally significant, features, such as the action *en masse* of other ocular muscles. One usually sees loss of superior and inferior rotation, due to simultaneous activation of the respective rectus muscles, as was demonstrated experimentally in the monkey by Bender and Fulton.[5] In the case reported here, vertical movements of the eyes both up and down were possible, although they were weak. The absence of diplopia may be accounted for by imperfect vision in the left eye. As is usually the case, the pupil of the affected eye was the larger; a light reflex may or may not be present, and in this case it was.

Pathologic Considerations: The pathologic changes in the roots of the oculomotor nerve have not to my knowledge been reported heretofore in this condition. The outstanding lesions were clearly neuromatous. The cell type within these areas contained large, plump, sausage-shaped nuclei and long cytoplasmic processes. The cells were collected in interlacing fascicles with palisaded nuclei. Each cell was surrounded by a collagenous endoneurial sheath, in which only a relatively small amount of longitudinal reticulin fibers could be specifically impregnated. On the other hand, there was an abundant fiber component, consisting of axons which, for the most part, were thin and sufficiently irregular to be considered regenerative unmyelinated fibers. According to Nageotte's views,[6] the cellular constituents of the lesions comprise elements of a Schwannian "syncytium" which have caught and enclosed the regenerating neurites. This was particularly clear in the cross-sectional views, in which axis cylinders were seen penetrating the delicate fenestrated tissue formed by tubular elements, the sheaths of which were separated one from another by a delicate collagenous framework.

The histologic changes were, I believe, similar to those described by Masson[7] and Holmes and Young[8] in experimental regeneration neuromas. The fasciculated, interlacing structure of the lesions in my case was possibly due to the proliferation of the regenerating tissue within the confines of the epineurial sheath, in contrast to experimental lesions, in which this limiting membrane is interrupted when the nerve is sectioned, allowing the exuberant tissue to be less restrained. The similarity of the cell aggregations in the spontaneous neurinomas ("aneuritic neuroma" of Masson) to those occurring in the course of regeneration, as in this case ("neuritic neuroma") may be more than a coincidence, and perhaps argues in favor of the Schwannian hypothesis of their nature. However, the common cell type has been identified variously as either a Schwann cell or a fibroblast. The divergence of these views concerns not only spontaneous tumors but also regener-

5. Bender, M., and Fulton, J. F.: Factors in the Functional Recovery Following Section of the Oculomotor Nerve in Monkeys, J. Neurol. & Psychiat. **2**:285, 1939.

6. Nageotte, J.: Sheaths of the Peripheral Nerves: Nerve Degeneration and Regeneration, in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, Vol. 1, Sect. 5, p. 191.

7. Masson, P.: Experimental and Spontaneous Schwannomas (Peripheral Gliomas), Am. J. Path. **8**:367, 1932.

8. Holmes, W., and Young, J. Z.: Nerve Regeneration After Immediate and Delayed Suture, J. Anat. **77**:63, 1942.

ation neuromas. Thus, Masson [7] presented an attractive hypothesis in favor of the Schwann cell, while Denny-Brown [9] offered cogent arguments for the neural fibroblast. As Percival Bailey concluded, after an extensive study of the problem, there are as yet no specific and reliable histologic methods for identifying Schwann cells.[10] Observations on the nerve changes in other cases of aneurysm and similar expanding processes and in experimental nerve lesions produced by progressive stretch will undoubtedly add to and clarify our knowledge of the histogenesis of nerve tumors.

One wonders why the neuromas extended proximally from the aneurysm and so close to the points of exit of the nerve roots from the neuraxis. Ramón y Cajal [11] observed in his spinal-nerve lesions that "a large number of the sprouts formed in the central stump of nerves . . . become retrograde." This pattern of regeneration seems less likely here than Cajal's "collateral neoformation" of nerve fibers, which occurs when a nerve is more diffusely damaged. The observation was made in the present material that neuromas formed only in the peripheral segment of the nerve roots, whereas in the glial segment the alterations of nerve fibers were mostly regressive in nature. The central segment of the nerve roots thus seemed incapable of true repair of axonal damage, with only a slight cellular reaction as its response to injury. This finding is, of course, in full accord with the general principle of the inability of the central nervous system (including the glial segments of nerves) to regenerate.

The clinical and pathologic findings in this case are interpreted on the basis of subchronic injury to the oculomotor nerve, setting up reparative (regenerative) response in those parts of the nerve trunk which possess such powers. The aneurysm, which was rather sizable, probably caused the injury through a mechanism of progressive stretch of the oculomotor nerve. The sparing of some of the nerve bundles is interesting, and is taken to be the reason that the functions of the nerve were not sufficiently altered as to produce the full picture of the pseudo-Graefe phenomenon.

Is there an alternative to the misdirection theory? In the course of an extensive review, Wartenberg [12] raised a series of objections to the hypothesis of misdirection of regenerating nerve fibers as the explanation of the pseudo-Graefe phenomenon and the comparable postparalytic facial hemispasm. Some of his arguments dealing with histopathologic processes deserve consideration in view of the present observations of a profuse neuritic overgrowth. First, it may be noted that in this case extensive regenerative changes took place in an oculomotor nerve which was the site of a nonsurgical injury never sufficiently complete to have produced the clinical picture of third-nerve paralysis. Thus, associated movements may occur in a regenerating nerve in which the nerve bundles have not been grossly distorted. A similar process could account for the appearance of hyperkinesis at a distance from an injured peripheral branch of the facial or oculomotor nerve. Retrograde spread of regenerating fibers to a main branch and sidetracking into uninjured branches are not conjectural, but were demonstrated by Ramón y Cajal.

9. Denny-Brown, D.: Importance of Neural Fibroblasts in the Regeneration of Nerve, Arch. Neurol. & Psychiat. **55**:171 (March) 1946.

10. Bailey, P., and Herrmann, J. D.: Rôle of the Cells of Schwann in the Formation of Tumors of the Peripheral Nerves, Am. J. Path. **14**:1, 1938.

11. Ramón y Cajal, S.: Degeneration and Regeneration of the Nervous System, London, Oxford University Press, 1928.

12. Wartenberg, R.: Associated Movements in the Oculomotor and Facial Muscles, Arch. Neurol. & Psychiat. **55**:439 (May) 1946.

In his classic studies on nerve regeneration, Cajal [13] showed that such retrograde fibers bifurcate repeatedly, grow indiscriminately, and travel through neurol interstices, even invading the white matter of the neuraxis. Howe, Tower, and Duel [14] found branching of regenerating fibers following interruption of the facial nerve in monkeys with postparalytic facial tic. Associated movements following peripheral-nerve injury are not confined to the territories of the facial and oculomotor nerves but occur also in muscles supplied by spinal nerves, as in the movements of the upper extremity associated with respiration recently observed by Robinson.[15] Congenital cases of associated movements of the eyes or face are rather more difficult to explain and may well comprise a different category of disorder. However, until we have pathologic data on these cases, we can only substitute conjecture. It is curious that hitherto such data in this field have been almost nonexistent. The only previous anatomical study of a case of pseudo-Graefe phenomenon with which I am familiar is that by Köppen [16] in 1894. Köppen found prominent and thickened blood vessels in the periaqueductal gray matter in his case but no change in the nerve cells or tracts, and he made no mention of the nerve roots; it is to be noted that the report was made before Lamy [17] and Lipschitz [18] offered the hypothesis of misdirected regenerating fibers to explain the comparable postparalytic facial hyperkineses.

As an alternative to the theory of fiber misdirection, Wartenberg [12] proposed that the retrograde cell degeneration of the cranial-nerve nucleus following peripheral injury sets up a dedifferentiation of a "frail, phylogenetically young superstructure controlling fine, isolated movements," which results in the appearance of mass movements in the cranial-nerve field. However, the axonal reaction should be looked upon as a self-limited process, to be reversed when the nerve injury has been repaired and the fiber component reconstituted. When the injury cannot be repaired, as in interruption of central nerve fibers, the chromatolytic process subsides, and the cell is transformed into one with a correspondingly short axon; the retrograde degeneration, as Cajal [19] pointed out, is propagated in a centripetal direction only up to the last bifurcation or collateral of the axon. The neuronal changes should be viewed as an adjustment to shortening of the axon, rather than as a true "degeneration." Transsynaptic retrograde degeneration in a higher, regulatory system appears highly improbable.

The prevailing view of faulty regeneration of peripheral nerve fibers is much simpler and is substantiated by such observations as are available at present. The very permanency of the associated movements argues for a structural change in the nerve trunk rather than for a functional reorganization of nuclear and supranuclear interrelations induced by temporary alterations of segmental motor neurons. Furthermore, interaction between somatic and autonomic components could hardly result from damage to a suprasegmental controlling mechanism, whereas it is readily accounted for by misdirection of regenerating fibers, as was pointed out by Ford, Walsh, and King [4] for the pupillary reaction in adduction.

One other feature of this case will be mentioned here but is discussed further in relation to the second case. Degeneration, gliosis, and accumulation of hematogenous pigment within phagocytes and astrocytes were observed in the cerebral cortex and subcortex of the temporal lobe adjacent to the aneurysm. These alterations were obviously due to compression and laceration by the hemorrhages resulting from previous ruptures of the aneurysm.

13. Ramón y Cajal,[11] p. 584.

14. Howe, H. A.; Tower, S. S., and Duel, A. B.: Facial Tic in Relation to Injury of the Facial Nerve: An Experimental Study, Arch. Neurol. & Psychiat. **38**:1190 (Dec.) 1937.

15. Robinson, P. K.: Associated Movements Between Limb and Respiratory Muscles as a Sequel to Brachial Plexus Birth Injury, Bull. Johns Hopkins Hosp. **89**:21, 1951.

16. Köppen, M.: Beiträge zur pathologischen Anatomie und zum klinischen Symptomencomplex multipler Gehirnerkrankungen, Arch. Psychiat. **26**:99, 1894.

17. Lamy, H.: Note sur les contractions "synergiques paradoxales" observees à la suite de la paralysis faciale périphérique, Nouv. iconog. Salpêtrière **18**:424, 1905.

18. Lipschitz, R.: Beiträge zur Lehre von der Facialislähmung nebst Bemerkungen zur Frage der Nervenregeneration, Monatsschr. Psychiat. u. Neurol. (Supp.) **20**:84, 1906.

19. Ramón y Cajal,[11] p. 665.

INTRACEREBRAL HEMORRHAGE

CASE 2.[20]—J. R., a white man aged 57, had had recurrent headaches since youth. These were generalized and unassociated with ocular complaints and were relieved after he vomited and lay down. In recent years the attacks had been less severe.

On Aug. 6, 1948, he had a sudden onset of generalized headache and vomiting and soon lost consciousness. A physician was called and found that the left arm dropped more quickly than the right. At Baylor University Hospital, the cerebrospinal fluid was found to be grossly bloody, later clearing to xanthochromic; and three weeks later it was colorless. At first he was restless, irrational, and poorly oriented. He moved the right arm but not the left arm or leg. This condition gradually improved; after five days, he became clearer mentally, and stated that he had no recollection for events since the onset.

A second hemorrhage occurred on Sept. 2, 1948, when he complained of headache and then collapsed. He was unresponsive and moved the extremities aimlessly; he vomited after a half-hour and soon regained consciousness. He was readmitted to the hospital for 3½ weeks, and during most of this period he complained of severe headache. At times he was irrational and disoriented for time, place, and person. Memory for recent events was poor. He was restless, especially at night. The left side of the face was slightly weak. The extremities were equal in strength on the two sides, but the abdominal reflexes were absent on the right, with a Babinski sign on that side. The cerebrospinal fluid was again grossly bloody, later gradually clearing. Compression of the left carotid artery was done preparatory to arteriography, but during this procedure he lost consciousness and manifested clonic movements of the right leg; on release of compression he immediately regained consciousness and resumed talking. This procedure was repeated several times during the stay in the hospital, always producing giddiness or unconsciousness, and so it was decided to forego angiographic studies. On discharge, he was still disoriented slightly, although at times he responded normally to questions. For about three months more he remained easily confused, and his wife stated that he never fully recovered his memory for recent events.

He was admitted to the Veterans Administration Hospital at Dallas in August, 1949, because of pain in the right hip region, which had been present for two years. He was well oriented. Some observers thought there was a slight paresis of the right side, with an increase in the reflexes and with elicitation of Hoffmann and Babinski signs on that side. Electroencephalography revealed low-voltage slow activity at a frequency of 3 and 7 per second in the right frontal region with spread to the left frontal region on a background of dominant alpha rhythm of 9 to 10 per second, the focal slowing persisting during sleep. Stimulation of the left carotid sinus produced cardiac slowing, impaired consciousness, and focal convulsive movements on the right side of the body, whereas massage of the right carotid sinus resulted only in slight slowing of the heart rate, without change in consciousness or any neurologic signs. Systemic studies, including biopsy of the right ilium, demonstrated an undifferentiated carcinoma (probably bronchogenic), with widespread metastases. He died in October, 1950.

Autopsy (by Dr. C. A. Johnson) disclosed a bronchogenic carcinoma with extensive hematogenous and lymphatic spread, including a microscopic metastasis in the posterior lobe of the hypophysis. Arteriosclerosis was marked in the aorta and moderate in the coronary and larger cerebral arteries.

Examination of the brain revealed that the leptomeninges were slightly thickened at the vertex of both cerebral hemispheres and contained numerous punctate areas of reddish-orange tint along the superior longitudinal fissure in the prefrontal region and, to a less extent, in the occipital region. The basal arteries presented a saccular aneurysm, 7 mm. in diameter, in the region of the anterior communicating artery (Fig. 7*A*). The circle of Willis was anomalous in that essentially the entire anterior cerebral circulation was derived from the left internal carotid artery. The left carotid artery was wider (3 mm.) than the right (2 mm.), and its walls were rather thicker. The left anterior cerebral artery in its primary, or horizontal, portion (between the internal carotid and anterior communicating arteries) was distinctly enlarged (2.5 mm. in diameter), whereas the corresponding vessel on the right was thread-like (0.4 mm. in width). The left anterior cerebral artery, in effect, bifurcated in the region where one expects to find

20. Dr. George M. Jones supplied clinical data on this case.

the anterior communicating artery, giving rise to right and left anterior cerebral arteries of equal caliber (2 mm.). The middle cerebral arteries were equal in caliber (2.5 mm.). The posterior communicating arteries were asymmetric, the right being normal in width (1 mm.), whereas the left was narrow (0.6 mm.). The basilar and posterior cerebral arteries were normal.

The aneurysm was bound down by moderately dense adhesions to the posterior orbital cortex within the sagittal fissure, pointing more toward the right side. On section the inferior medial sector of the right frontal lobe was found to be occupied by a cyst, the lining of which was smooth and stained a rust color (Fig. 7*B*). The cyst was largely subcortical, but it involved the cortex in the posterior orbital region, narrowing it to a width of 1 mm. It extended from the frontal pole posteriorly to the anterior commissure and superiorly to the base of the frontal horn, where the ependyma had a rusty discoloration. The inferior one-third of the septum pellucidum was degenerated on the right side back to the anterior commissure.

On microscopic examination the lining of the cyst was observed to consist of loose tissue, containing large numbers of protoplasmic astrocytes and phagocytes. In all areas of the cyst wall the phagocytes were loaded with hemosiderin, and a small amount of this pigment was also

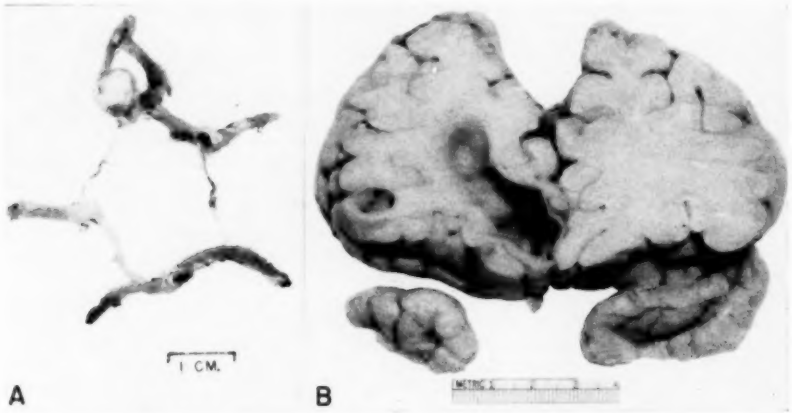

Fig. 7 (Case 2).—*A*, basal cerebral arteries, with multiple anomalies; saccular aneurysm in region of anterior communicating artery. *B*, coronal section of frontal lobes, showing cyst in medial basal portion of right frontal lobe and metastatic carcinomatous nodule in inferior frontal gyrus.

contained within the cytoplasm of large astrocytes. In places there were accumulations of fat-filled phagocytes. Silver impregnation of the cyst wall showed a delicate reticulin framework. In the involved portion of the cerebral cortex groups of ganglion cells had undergone iron encrustation. Hemosiderin was noted in macrophages within the pigmented areas of the leptomeninges and also scattered through these membranes at the base in the area of the aneurysm and as far posteriorly as the hindbrain; the basal surface of the right frontal horn likewise showed phagocytosed blood pigment in densely sclerosed subependymal tissue. In the superficial layers of the cerebral cortex, blood pigment was included within hyperplastic microglial elements and small astrocytes.

The aneurysmal wall was composed of compact collagenous connective tissue, the outer layers of which contained scattered phagocytes filled with fat, as well as granular and amorphous calcific material. The lining of both lateral ventricles and the third ventricle presented granular proliferations of subependymal glia; this process was most intense in the right lateral ventricle near the anterior commissure. The leptomeninges were moderately fibrosed. About two dozen small metastatic nodules were scattered throughout the brain, being unconnected with the cyst in the frontal lobe.

Comment.—It is clear that hemorrhage from the saccular aneurysm in this case occurred into the medial basal portion of the right frontal lobe, as well as into the subarachnoid space. The clinical manifestations during the episodes of intracranial bleeding suggest that the hemorrhage into the cerebral substance occurred during the first attack, in which transient left hemiplegia was an early sign. The confusion and disorientation noted in both episodes, severer and more enduring in the second, were possibly due to focal involvement of basofrontal radiations. Richardson and Hyland [21] pointed out the prominence of intellectual retardation in cases in which frontal-lobe hemorrhage resulted from rupture of aneurysm on the anterior cerebral, anterior communicating, or middle cerebral artery.

In the second attack of hemorrhage, minimal signs of right hemiplegia (absence of abdominal reflexes, presence of the Babinski sign) suggested involvement of the left cerebral hemisphere as well; yet at necropsy there was no evidence of a vascular lesion in this region. In view of the fact that the aneurysm was situated actually at the bifurcation of an anomalous primary segment of the left anterior cerebral artery, which then gave origin to both anterior cerebral arteries, it is not difficult to visualize a disordered circulation in the territory of the cortical or subcortical branches of the left anterior cerebral artery. In some cases of aneurysm with cerebral signs, absence of intracerebral hemorrhage at autopsy indicates that other mechanisms may on occasion be responsible for focal cerebral signs. Ischemic foci may be caused by thrombosis of the parent vessels (Garvey [22]; Jefferson [23]; Courville and Olsen [24]), peripheral emboli from a thrombus within the aneurysm (Biemond and ter Braak [25]), or, probably not infrequently, vasomotor influences in the portion of the brain supplied by the artery, or even in remote areas (Robertson [26]). Cerebral edema may be the only parenchymatous change, such as was noted by Doyle [27] and also was found in a personally observed specimen from a case of subarachnoid hemorrhage and left hemiplegia, with demonstration at autopsy of an aneurysm of the right anterior cerebral artery and diffuse edema of the right cerebral hemisphere.

In cases of intracerebral hemorrhagic foci secondary to saccular aneurysms, the lesions heretofore reported have been relatively fresh. The recent character of the lesions is largely due to the rather high mortality occurring in the presence of this complication, so that the pathologic changes are those of recent hemorrhage. Or, when recovery from intracerebral hemorrhage does take place, the changes are likely to be concealed by a recurrent, and fatal, extravasation. This was found to have occurred in Case 1 of this paper. In Case 2 it was pure chance which led to death from a disease which in no way obscured the healed lesions of intracerebral

21. Richardson, J. C., and Hyland, H. H.: Intracranial Aneurysms, Medicine **20**:1, 1941.

22. Garvey, P. H.: Aneurysms of the Circle of Willis, Arch. Ophth. **11**:1032 (June) 1934.

23. Jefferson, G.: Compression of the Chiasma, Optic Nerves, and Optic Tracts by Intracranial Aneurysms, Brain **60**:444, 1937.

24. Courville, C. B., and Olsen, C. W.: Miliary Aneurysm of the Anterior Communicating Artery, Bull. Los Angeles Neurol. Soc. **3**:1, 1938.

25. Biemond, A., and ter Braak, J. W. G.: Über die sogenannten spontanen subarachnoidalen Blutungen und ihre Beziehung zum Aneurysma der Hirngefässe, Deutsche Ztschr. Nervenh. **132**:22, 1933.

26. Robertson, E. G.: Cerebral Lesions Due to Intracranial Aneurysms, Brain **72**:150, 1949.

27. Doyle, J. B.: Spontaneous Subarachnoid Hemorrhage, California Med. **73**:336, 1950.

and subarachnoid hemorrhage. That the process in this case was one of hemorrhage, rather than infarction, is indicated by the pigmentation of the entire lining of the cyst. Moreover, the location of the lesion in the medial and basal sector of the frontal lobe is the characteristic position of hemorrhage from aneurysms in the region of the anterior communicating artery (Courville and Olsen [24]), in contradistinction to the widely separated distribution of malacic foci in thrombosis of the anterior cerebral artery.

The only instance of hemorrhagic cyst in cases of subarachnoid hemorrhage that I have encountered in the literature is one reported by Ingvar.[28] In this case, right hemiplegia occurred during an acute cerebral disorder (hemorrhage?) at the age of 14 years, with gradual recovery, only to be followed by the appearance of nocturnal generalized convulsions. The patient died at the age of 24 of extensive subarachnoid hemorrhage. At autopsy, in addition to the leptomeningeal effusion, a cyst with a brown lining was encountered in the left corpus striatum.

In one of the subjects tested by Davis [29] and his collaborators for the uptake of radioactive diiodofluorescein a lesion of the right frontal lobe was demonstrated by electroencephalography, ventriculography, and angiography which at operation was proved to be an aneurysm of the anterior cerebral artery with multiple areas of old intracerebral hemorrhagic softening; further details of this case have not been published.

The anomaly of the circle of Willis was undoubtedly a factor in the pathogenesis of the saccular aneurysm. This has been noted previously, especially by Slany [30] and more recently elaborated by Padget.[31] The blood flow within the anomalous vessels may produce undue stress at the bifurcations, and if the arterial media happens to be defective at this point aneurysmal formation will in time result.

SUMMARY

Late neuropathologic changes in cases of intracranial saccular aneurysms are described.

In the oculomotor nerve, regeneration neuromas occurred in a case in which the pseudo-Graefe phenomenon was presented. This sign developed without nerve paralysis ever having appeared in the usual form. The histologic changes provided confirmation of the theory of misdirection of regenerating nerve fibers as the basis of postparalytic associated movements.

Residual lesions of previous intracerebral hemorrhage were demonstrated in two cases. In one, the changes were uncomplicated by recent hemorrhage, the patient having died of coincidental carcinomatosis.

28. Ingvar, S.: Sur les hémorrhagies méningées, Nouv. iconog. Salpêtrière **28**:313, 1916-1917.

29. Davis, L.; Martin, J.; Ashkenazy, M.; LeRoy, G. V., and Fields, T.: Radioactive Diiodofluorescein in Diagnosis and Localization of Central Nervous System Tumors, J. A. M. A. **144**:1424 (Dec. 23) 1950.

30. Slany, A.: Anomalien des Circulus arteriosus Willisi in ihrer Beziehung zur Aneurysmenbildung an der Hirnbasis, Virchows Arch. path. Anat. **301**:62, 1938.

31. Padget, D. H.: The Circle of Willis, Its Embryology and Anatomy, in Dandy, W. E.: Intracranial Arterial Aneurysms, Ithaca, Comstock Publishing Co., Inc., 1944, Sect. 3, p. 67.

PSYCHOLOGICAL FUNCTIONING FOLLOWING CEREBRAL HEMISPHERECTOMY IN MAN

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THE PSYCHOLOGICAL effects of cortical destruction in man have been recognized for many years. Even the effects of damage to an area as extensive as an entire hemisphere had been described decades ago, as Karnosh and Gardner [1] have noted.

Over a hundred and fifty years ago, Francis Joseph Gall presented a case report on a man in whom the right hemisphere had been totally destroyed by suppuration and yet the intellectual faculties had remained intact up to the time of the death.

Much of our knowledge of the effects of decortication has come from animal operations, for example, Kellogg's [2] long-time studies at Indiana University. Even earlier, Karplus and Kreidl [3] had accomplished decerebration in animals, including apes. It was not until 1923 that Dandy [4] performed the first hemispherectomy on man. Since his first published report in 1928, a number of articles in the literature have dealt with hemispherectomy. Some have been general discussions of the operative technique[5]; others have summarized special studies of postoperative functioning, e. g., Bunch [6] on auditory acuity, Walker [7] on pain pathways, and Zollinger and Schnitker [8] on skin temperature. A survey of the medical, psychiatric, and

Mrs. Barbara Richman assisted in this study.

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1. Karnosh, L. J., and Gardner, W. J.: Physical and Mental Capacity After Removal of a Right Cerebral Hemisphere, Dis. Nerv. System **1**:343-348, 1940.

2. Kellogg, W. N.: Conditioning Involving the Two Body Sides After Hemidecortication, Am. J. Psychol. **3**:237, 1948.

3. Lereboullet, J.: Removal of Left Cerebral Hemisphere, Paris méd. **1**:358-360, 1936.

4. Dandy, W. E.: (*a*) Removal of Right Cerebral Hemisphere for Certain Tumors with Hemiplegia, J. A. M. A. **90**:823-825 (March 17) 1928; (*b*) Physiological Studies Following Extirpation of the Right Cerebral Hemisphere in Man, Bull. Johns Hopkins Hosp. **53**:31-51, 1933. Lereboullet.[3]

5. Lereboullet.[3] Lhermitte, J.: Complete Excision of the Right Hemisphere, Encéphale **23**:314-323, 1928.

6. Bunch, C. C.: Auditory Acuity After Removal of Entire Right Cerebral Hemisphere, J. A. M. A. **90**:2102 (June 30) 1928.

7. Walker, A.: Central Representation of Pain, A. Res. Nerv. & Ment. Dis., Proc. **23**:64-85, 1943.

8. Zollinger, R., and Schnitker, M. T.: Skin Temperature Reactions Following Removal of Left Cerebral Hemisphere, Science **79**:540, 1934.

psychological literature published since 1928 yielded reports on 41 hemispherectomies, 28 of which were done in this country, including Dandy's early series of 7. A summary of the cases reported is presented in Table 1.

With few exceptions, the preoperative and postoperative psychological functioning of the patients has been described in general terms. Thus, the reports of the psychological state before operation have consisted of such notes as the following:

. . . marked emotional disturbance.[9]

TABLE 1.—*Cerebral Hemispherectomies Performed from 1923 to 1950*

Age	Sex	Date of Operation	Hemisphere	Survival Period	Author
..	M	1923	Right	3½ yr.	Dandy, 1928
..	M	....	Right	3 mo.	Dandy, 1928
..	M	....	Right	2 days	Dandy, 1928
32	M	1923	Right	2 yr.	Dandy, 1933
35	F	1927	Right	2 mo.*	Dandy, 1928
..	M	1927	Right	2 wk.	Dandy, 1928, 1933
24	F	1927	Right	6 mo.	Dandy, 1933
31	F	1931	Right	4½ yr.	O'Brien, 1932, 1936
43	F	1933	Left	17 days	Zollinger, 1934, 1935
38	F	1936	Right	6 mo.*	Rowe, 1937
..	..	....	.....	.....	Gardner, 1932, 1933
..	..	....	.....	.....	Karnosh and Gardner, 1941
..	..	....	Right	1½ days	Gardner, 1933
..	..	....	Right	1½ days	Gardner, 1933
25	F	....	Right	29 days	Karnosh, 1937; Karnosh and Gardner, 1941
..	..	....	Right	15 mo.	Karnosh and Gardner, 1940
27	M	....	Right	2½ yr.*	Karnosh and Gardner, 1941
35	M	1938	Right	10 yr.*	Karnosh and Gardner, 1941
..	..	....	.....	.....	Bell and Karnosh, 1949
		(3 other patients operated on; no further data reported)			
..	M	1941	Right	1 yr.	Walker, 1943
9	F	1945	Left	2 yr.*	Krynauw, 1950
21	F	1948	Left	1 yr.*	Krynauw, 1950
		(10 other patients operated on; no further data reported)			
51	M	1949	Right	9½ mo.*	Marshall and Walker, 1950
64	M	1949	Right	5 mo.	Marshall and Walker, 1950
11	M	1949	Right	7 wk.*	Marshall and Walker, 1950
23	F	1949	Right	2 mo.*	Marshall and Walker, 1950
18	F	1949	Right	3 mo.*	Obrador and Larramendi, 1950
37	M	1949	Left	4 mo.	Crockett and Estridge, 1951
54	M	1950	Right	17 days	Crockett and Estridge, 1951
56	M	1950	Right	2½ mo.	Crockett and Estridge, 1951
67	M	1950	Right	21 days	Crockett and Estridge, 1951
54	M	1950	Right	6½ mo.	Mensh, Schwartz, and Matarazzo (present report)

* Patient still living at time of report.

. . . unreasonable and unmanageable temper, lacked power to concentrate . . . mental lapses . . . institutionalized because temper tantrums and irritability made long discipline impossible. Lately had 1-2 minute spells of nonsense, uncontrolled shouting. . . . Mentally slow, could not read or write. . . . Unhappy, depressed, and acutely aware of disabilities . . . afraid to mix with people. . . . Disregard for personal appearance . . . felt she had nothing to live for.[10]

9. O'Brien, J. D.: Removal of the Right Cerebral Hemisphere: Further Report on Case, J. A. M. A. **107**:657 (Aug. 29) 1936.

10. Krynauw, R. W.: Infantile Hemiplegia Treated by Removal of One Cerebral Hemisphere, South African M. J. **24**:539-546, 1950.

. . . irritability and a tendency to fight and show violent temper towards her relatives.[11] . . . brilliant, eccentric, seclusive . . . nervousness, agitation, complaining, depression, queer talk and rumbling, unresponsive . . . confabulated . . . fairly well oriented but had difficulty in maintaining intellectual contact.[4b]

. . . emotional instability . . . unable to cooperate with examiner.[12]

In equally general terms, postoperative psychological behavior has been reported. Although there has been agreement on the clinical postoperative observations, in only a few instances have data from objective psychological examination been included. The following clinical studies of postoperative behavior are characteristic:

. . . there did not seem to be any obvious mental impairment . . . we could not detect any mental impairment from these cursory tests. . . .[4a]

. . . The presentation of these three cases with extirpation of such vast areas of brain tissue without the disclosure of any resulting defect in mental functioning is most disappointing. . . . I am still not willing to say that the mentality of the patients was normal, but rather that abnormalities have not been disclosed. . . .[4b]

. . . There was no demonstrable intellectual impairment. . . . The preservation of function may be quite remarkable . . .[13]

. . . Post-operative tests of physical and mental capacity in most of the patients, who live long enough to survive the shock and infection secondary to such a formidable procedure, have shown relatively little dementia considering the degree of brain mutilation . . .[1]

. . . the relative freedom from severe physical handicap and gross mental defect after removal of almost half the cerebrum was striking. . . . Penfield (1934) stated that the defect produced by a frontal lobectomy is a lack of capacity for planned administration and the loss of power of initiative. Study of our cases has indicated that this is the most accurate description of the mental defect produced by removal of the right cerebral hemisphere . . .[14]

More specific reference to psychological studies of postoperative behavior is made by Karnosh and Gardner.[15] They summarized their findings by reporting:

Mental changes extremely elusive . . . No pathologic disorientation . . . wide swings in feeling tone. . . . Positive tests produced no striking abnormalities. Greatest difficulty lay in inability to concentrate, to deliberate at great length and to carry out concerted, protracted mental procedure.

Krynauw[10] operated on a woman aged 21 whose preoperative mental age was 10 years 4 months, but the postoperative level was not reported, although the general psychological improvement was pronounced. Similarly, Obrador and Larramendi[11] summarized their study of a girl aged 18: "Mental tests show a post-operative

11. Obrador, S., and Larramendi, M. H.: Some Observations on the Brain Rhythms After Surgical Removal of a Cerebral Hemisphere, Electroencephalograph. & Clin. Neurophysiol. **2**:143-146, 1950.

12. Zollinger, R.: Removal of Left Cerebral Hemisphere, Arch. Neurol. & Psychiat. **34**:1055-1064 (Nov.) 1935.

13. Gardner, W. J.: Removal of the Right Cerebral Hemisphere for Infiltrating Glioma, J. A. M. A. **101**:823-826 (Sept. 9) 1933; abstracted Arch. Neurol. & Psychiat. **28**:470 (Aug.) 1932.

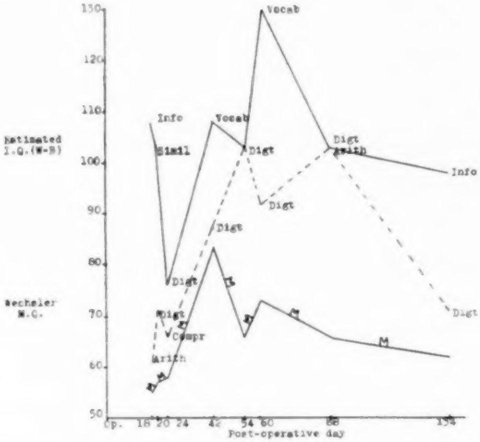
14. Bell, E., and Karnosh, L. M.: Cerebral Hemispherectomy: Report of a Case Ten Years After Operation, J. Neurosurg. **6**:285-293, 1949.

15. Karnosh, L. J., and Gardner, W. J.: An Evaluation of the Physical and Mental Capabilities Following Removal of the Right Cerebral Hemisphere, Cleveland Clin. Quart. **8**:94-106, 1941; footnote 1.

improvement in memory." Dandy also reported psychological studies of his patients when these were possible. About two of his patients he commented:

. . . always perfectly oriented as to time, place and person. . . . Their memory for immediate and remote events was unimpaired. They could read, write and compute without error.[4a]

Finally, Rowe[16] was interested principally in the psychiatric aspects of postoperative behavior and included psychological tests in his study of a woman aged 38. The Stanford-Binet Intelligence Test was administered before operation, four months after operation and, again, after two months, a total of three intellectual evaluations within a period of about a year. Of above-average intelligence, with an intelligence quotient of 115, the patient "showed a slight improvement" in the second and third postoperative tests.



Psychological functioning following cerebral hemispherectomy. Peak intellectual functioning is indicated by a solid line; poorest functioning, by a broken line, and memory performance, by the solid line with *M*.

PSYCHOLOGICAL AND NEUROLOGICAL DATA

In none of the published reports of psychological functioning following hemispherectomy has there been a systematic study with standard, objective techniques. In the case to be discussed, the Wechsler Memory Scale, Form I,[17] was used as the principal instrument, and this was supplemented at intervals with the Wechsler-Bellevue Intelligence Scale, Form I, and the Rorschach test.[18] The psychological

16. Rowe, S. N.: Mental Changes Following Removal of Right Cerebral Hemisphere for Brain Tumor, Am. J. Psychiat. **94**:605-612, 1937.

17. Wechsler, D.: A Standardized Memory Scale for Clinical Use, J. Psychol. **19**:87-95, 1945.

18. Beck, S. J.: Rorschach's Test II, New York, Grune & Stratton, Inc., 1947. Ulett, G. A.: Rorschach Workbook, St. Louis, Washington University School of Medicine, 1950; Rorschach's Test as Used in the Psychiatric Interview, Northwest. Med. **48**:544-548, 1949.

examinations were carried out on postoperative days 19, 20, 24, 42, 54, 60, 88, 96, and 134. The Wechsler Memory Scale and various subtests of the Wechsler-Bellevue Intelligence Scale were administered on each occasion, and the Rorschach test was given on the 95th postoperative day. The patient began to have a series of convulsions during the 6th postoperative month and died after 2 weeks, 6½ months after the operation.

The Wechsler Memory Scale is designed to sample psychological functioning in the areas of personal and current information (age, date of birth, President of the United States, governor of home state of patient, etc.), orientation to time and place, mental control with speed and accuracy of function measured, immediate recall of logical material presented verbally in a passage of several sentences, rote memory (digit span), and paired association (the learning of paired words with later recall as a test of degree of learning). Visual-motor organization and coordination as measured by reproduction from memory of geometric designs are also sampled, but in our patient this area could not be tested because of postoperative hemiplegia and visual difficulty. The motor difficulties of the patient also precluded the use of performance tests; therefore, only the verbal material of the Wechsler-Bellevue Scale could be used. This included items sampling information and rote memory (as in the Wechsler Memory Scale, but containing more items), comprehension, arithmetical ability, similarities (abilities to form concepts and to derive abstract relationships), and vocabulary. The Rorschach test is a projective technique widely used in personality study, an instrument utilizing the perceptual reactions of the subject as a basis for study of the personality structure.

Four days before operation, the patient, a white man aged 54, had been admitted to Barnes Hospital because of increased severity of headaches (history of band-like headaches since the age of 14, when the patient had malaria), insomnia, irritability, loss of mental alertness, poor memory, weakness of the left side, and blurring of vision. At admission, the patient was noted to be lethargic, oriented, monosyllabic, and incomplete in his responses to questions, slow and halting in speech, but without aphasia or speech disorder. The neurological examination indicated an intracranial space-occupying lesion, and ventriculographic examination four days after his admission to the hospital was followed by a right frontoparietal craniotomy, with removal of the right cerebral hemisphere for tumor. The following data are abstracted from the operative and neuropathological notes. The family refused permission for necropsy; therefore no necropsy information is available.

"On admission the patient was lethargic, but on being roused was well oriented. Answers to questions were rather slow and monosyllabic. The patient was right-handed, but two of his siblings were left-handed. The left pupil appeared to be slightly larger than the right. Neurological examination revealed bilateral early papilledema, weakness of the left arm with awkwardness of finger movements on the left and a suggestion of a left grasp reflex, positive toe signs on the left, hypesthesia of the entire left half of the body to pinprick, and slight diminution of light-touch sensation. An electroencephalogram revealed dysrhythmia with a definite focus in the right parietal area. Roentgenograms of the skull revealed nothing abnormal.

"After his transfer to the neurosurgical service, operation was performed. Ventriculograms demonstrated a shift of the ventricular system to the left, and the anterior horn was lower on the right than on the left. An obvious defect jutted into the right lateral ventricle, just behind and superior to the foramen of Monro. In view of the brief history of symptoms and the probability of a malignant glioblastoma, deliberate resection of the right hemisphere was planned. This was carried out through a right frontoparietal craniotomy. At operation an extensive tumor, involving the posterior part of the frontal lobe and the anterior part of the parietal lobe, was demonstrated. Total right hemispherectomy was performed. The only neural material left on the right side was the thalamus. Examination of the specimen removed showed that a total hemispherectomy had been done, with removal of the lentiform nucleus. Tumor tissue

was noted in the lateral portion of the corpus callosum, but the genu of the internal capsule was clear. The tumor was a glioblastoma.

"During the first 48 hours after operation, the patient received adrenocortical extract to maintain his blood pressure at a level of about 110 mm. By the third day, the pressure rose to 140 mm. and was maintained at that level. For the first five days, his course was surprisingly good, and he responded well to commands. Four days after operation there was evidence of appreciation of pain on the hemiplegic side. Five days after operation he became drowsy, and this was found to be associated with increased nonprotein nitrogen of the blood. He also had an increase in temperature, and there was evidence of bronchopneumonia. This resulted in a rather stormy time for the next two weeks. Subsequently his condition improved slowly, and from that time the major difficulty consisted in trying to get him to be more alert and responsive. At first he tended to speak in a whisper, but finally, after considerable urging, this situation improved. At the time of discharge, the sense of smell was diminished on the right, the right disk could not be visualized, the left disk was pale, and there was left homonymous hemianopsia. Neither pupil reacted to light; the right eye could not be moved nasally. In tests for trigeminal sensation, the patient stated that the sensation appeared to be equal on the two sides, but objectively sensation was obviously greater on the right than on the left. There was paralysis of the left side of the face of central type. Hearing was good on both sides; no movement of the trapezius or sternocleidomastoid muscles could be detected on the left, and the tongue deviated to the left. No voluntary movement could be obtained in the left upper extremity; the left upper and lower extremities moved in response to painful stimulation, the reaction being interpreted as a reflex withdrawal; some voluntary contraction in the quadriceps muscles was demonstrable on the left. Pinprick could be appreciated on the left side, but was evidently not as acute as on the right; localization of sensation was poor on the left; astereognosis was present on the left. Position sense was impaired on the left side. Light touch appeared to be appreciated on both sides. Deep reflexes were increased on the left, and toe signs were elicited on the left. He had no aphasia and no difficulty in distinguishing between his left and his right side. It was attempted to obtain a detailed audiogram prior to his discharge, but the patient tired too easily.

"The final diagnosis was brain tumor (glioblastoma) in the right frontoparietal area. Ventriculography, followed by right craniotomy, with total right hemispherectomy, was performed.

"*Gross Pathologic Study.*[19]—The specimen consisted of the right cerebral hemisphere, submitted in three large blocks. The total specimen weighed 610 gm. The block of frontal lobe weighed 165 gm.; the middle block, 265 gm., and the occipital block, 180 gm. They were divided along the line just anterior to the middle part of the Sylvian fissure and another line just posterior to the angular gyrus. Over the cut surfaces on the medial portion of the specimen, there were included a portion of the corpus callosum and a ragged portion of the internal capsule, which lies lateral to the thalamus. It was not possible to identify exactly what structures were missing from the medial portion of this specimen, but apparently part of the hypothalamus and all of the thalamus were not present. There were scattered foci of subarachnoid hemorrhage in "step" sections through the brain. In all three specimens there were petechiae in the cortex, which contained 0.5 to 1.5 cc. of clotted blood. The frontal specimen contained the main tumor mass, which lay medially above the rostrum of the corpus callosum and extended to the cortical surface, above the medial and the superior portion of the superior frontal gyrus. The tumor at that point was about 3 cm. in diameter and was composed of a granular, gray, infiltrating tissue. Inferiorly and medially, there was a large focus of yellow necrosis, 1.5 cm. in its largest diameter. From this point the tumor extended posteriorly into the second part of the specimen, where it lay largely in a medial position, occupying the cingulate gyrus and infiltrating grossly into the corpus callosum. Back of the genu of the internal capsule there was no gross evidence of tumor, although a suspicious zone was noted on the medial portion of the lentiform nucleus. The approximate total measurements of the tumor were 3 by 3 by 6 cm., the largest measurement being in a vertical plane, from the frontal cortex to the inferior portion of the tumor at the level of the rostrum of the corpus callosum. Several sections were taken from the medial surface of

19. Dr. David E. Smith, of the Department of Pathology, Washington University School of Medicine, made available these data.

the specimen and fixed in Zenker's solution. A specimen was taken from the anterior portion of the corpus callosum, where grossly the tumor seemed to invade that structure. Another section was taken from the posterior portion of the corpus callosum, apparently free of tumor. A third specimen was taken from the medial portion of the lentiform nucleus at a plane perpendicular to the surface, that is, a coronal plane. A fourth specimen was a representative portion of the major tumor mass in the frontal lobe.

"*Microscopic Study.*—The tumor was a cellular mass composed of irregular astrocytes. There were slight pleomorphism and a few mitotic figures. There was marked proliferation of vessels, but only a few small foci of necrosis and pseudopalisading. The section from the anterior portion of the corpus callosum contained tumor, but sections from the posterior portion and medial surface of the lentiform nucleus were free of tumor. The tumor appeared to be derived from astrocytes; and, although many of the classic features of a glioblastoma multiforme were present to only a slight degree, the cellular density, mitotic figures, and pleomorphism indicated a speed of growth of the order of that tumor. The diagnosis was glioblastoma multiforme of the right cerebral hemisphere."

Psychological Studies.—On the 13th postoperative day the patient was seen for the first attempt for psychological examination. At this time he was drowsy and difficult to arouse, and testing could not be done. Three days later he again was seen for psychological study, but an acute infection of the respiratory tract with difficulty in breathing had necessitated his being placed in an oxygen tent, and again no examination of his psychological functioning could be done. On the following day his condition had improved sufficiently to permit his removal from the oxygen tent, but he slept most of the time, failed to respond except after much prompting, which produced only responses of "yes and no," and each such response was followed by apparent sleep.

On the next day, the 18th postoperative day, the patient seemed more alert than at any time since operation. He was able to use only his right arm and left eye, because of left hemiplegia and ptosis of the right eye. There were pronounced tremors of the hand and difficulty in turning the head and trunk, but the patient was able to fixate objects with his left eye at distances up to 18 in. (46 cm.). He spoke in a whisper, with occasional normal volume, and his voice generally was easily audible. In his responses to a test for aphasia and to portions of the Wechsler Intelligence and Memory Scales, he clearly showed superior vocabulary and verbal facility, a compulsive detailing of his responses, marked perseveration of ideas, and the psychological impotence often seen in brain-damaged patients when they apparently have the correct information but are unable to marshal and communicate it. He was able to identify a series of common objects, such as a miniature automobile, a doll, thimble, locomotive, cat, dog, and chair, and to give their uses, differentiating even color as significant when he named a toy automobile "a service car because it's yellow." He correctly manipulated a pair of scissors and a comb, followed directions in handling the various test objects, and used both tactual and visual cues to identify the objects. Thus, he responded to a toy shoe with "whistle," and later to an orange cube with "another variation of a cop's whistle"; after examining a toy cat tactually and visually, he sought for an auditory cue by holding it to his ear. Hearing no sound from it, he concluded that it was "a token for commercial use." His responses showed the compulsive detailing noted previously, and each object named was qualified—"a miniature cup . . . ordinary bottle . . . musical chair . . . plastic automobile."

Samples of the patient's responses are given below in illustration of the concreteness and perseveration of ideas, confused and psychotic-like thinking, clang associations, mingling of old and new information, and self-reference.

Vocabulary: P.O. 24[30]:
1. Apple—You buy it in the store. (?) Have it with sausages. (?) A round sphere.
5. Nuisance—Placing yourself in a position. (?) Doing an awful lot of things to society.

P.O. 42:
7. Cushion—Cloth or leather to maintain irregularities of the body.

P.O. 60:
27. Pewter—Metal used for cooking, handling hot liquids. They haven't used it for a long time, it's probably in short supply.
29. Catacomb—Graves of old patriarchs taken prisoners by the Romans.
30. Spangle—Metal ornament used to applique or spot on new garment, quite lively.
31. Espionage—Subterfuge applied to get information from an enemy, tactics not upright.
36. Dilatory—Very slovenly, improper tactics, don't follow rules of this guide.

Comprehension: P.O. 24:
2. Fire in theater—Probably have a fit.
3. Keep away from bad company—Only when we can't trust ourselves.
6. Land in a city—Costs more than land in the country; no one really knows.
7. Lost in a forest—Look for the North Star and get your bearings. (In daytime?) The same way, but it's not so easy to do.
8. Laws—Because people are selfish.
9. Marriage license—Keep society out of the mud.

Similarities: P.O. 20:
1. Orange, banana—Not native to here, tropical instead.
3. Dog, lion—One is crazy, I guess. I've got a quirk in my thinking.
4. Wagon, bicycle—Rubber-tired progress.
5. Daily paper, radio—They have to be up-to-date, and they give you what's represented.
7. Wood, alcohol—Exactly alike, for transmission and power.
8. Eye, ear—Both are normal reactions.
10. Poem, statue—Both have a thought meter. (?) It's like rhythm in music.
11. Praise, punishment—Human nature. (?) Thought meter. (?) Both have a definite meter of thinking.
12. Fly, tree—We're still crazy people.

P.O. 60:
6. Air, water—Free, about the only thing that is free any more.
10. Poem, statue—Try to depict a charm and grace of some fine character.
11. Praise, punishment—That question sounds screwy, nuts in other words.
12. Fly, tree—That one's got me, too.

Information: P.O. 18:
4. London—On the Thames, in England.
7. Italy—Don't know. I should.
10. Plane—Lorrain.
12. Paris-New York—650 miles.
14. Hamlet—Victor Herbert.
15. U. S. population—600 million.
16. Washington's birthday—August 22.
18. Egypt—South America.
19. H. Finn—Errol Flynn.
22. Faust—Rembrandt.
23. Habeas corpus—Writ setting forth the act of death or demise of a person according to record.

P.O. 60:
8. Japan—Florence.
10. Plane—Marconi.
12. Paris-N. Y.—1,000 miles.
15. U. S. population—approximately 500,000.
16. Washington's birthday—Feb. 22, unless Roosevelt changed it.
18. Egypt—South America.
21. Koran—A bible for those who believe in the Dramatic Order of the Knights of Korazan.
25. Apocrypha—5 or 6 chapters in the Old Testament. Somebody found 'em somewhere; they're not inspired.

The objective scores earned by the patient during the follow-up examinations are reported in Table 2, and in them the patient's variable postoperative psychological functioning is reflected.

TABLE 2.—*Postoperative Psychological Functioning*

	Postoperative Day							
Test Area	18*	20	24	42	54	60	88	134
Wechsler-Bellevue Scale								
Information	13†	..	..	..	..	16	..	11
Comprehension	..	..	3	..	..	..	..	..
Digits forward	4	5	6	6	7	6	7	5
Digits backward	2	2	2	3	4	3	4	2
Arithmetic	2	..	..	6	..	..	7	4
Similarities	..	11	..	..	..	8	..	..
Vocabulary	..	..	4	22	..	32	..	..
Wechsler Memory Scale								
Information	6	6	6	6	4	3	1	2
Orientation	2	3	3	3	2	0	0	2
Mental control	1	2	3	7	5	5	4	2
Logical memory	..	..	..	14	2	10	7	6
Digits forward	4	5	6	6	7	6	7	5
Digits backward	2	2	2	3	4	3	4	2
Associative learning								
Easy	..	..	..	4	6	8	8	10
Hard	..	..	..	1	3	4	3	1
Age correction (C. A.‡ 54)	44	44	44	44	44	44	44	44
Total memory score	59	62	64	86‡	74	79	74	69
Memory quotient	55	57	58	83	66	73	66	62

* Patient was unable to cooperate on postoperative days 13 to 17.
† Scores reported are raw scores.
‡ Total raw score for associative learning = (easy/2) + hard.
§ Chronological age.

20. "P.O." with a numeral indicates the postoperative day of the test.

In graphic form, the Chart presents a summary of the test data, and evident are the patient's fluctuations in efficiency of psychological function, the areas in which his best performance appeared, the areas in which he did most poorly, and the general level of his memory. The last is based upon the Wechsler Memory Scale, with its areas of personal and current information, orientation to time and place, mental control, immediate recall of logical material, digit span, and learning of paired words of both low and high association value.

Finally, the Rorschach test, a projective technique demanding visual perception, was administered on the 95th postoperative day. The protocol follows:

P.O. 95:

Card I:	107″	1. Mother Goose. (Ceuz of same thing that makes me look like me—head, wing in this case.) That's all. 3′32″
Card II:	31″	2. Looks like a camel but I never saw one dressed like that. (Head, neck, and general all-over look. A camel's hat—the design of it). 3′17″
Card III:	36″	Is it all there? 173″ 3. A crazy chipmunk, his head, neck, body. His body is all torn up. 4′37″
Card IV:	31″	4. Shades of Shakespeare—the albatross is not dead—from one of his stories. This is a vulture-like bird. (This is his head and his body). 2′34″
Card V:	19″	5. A wild party . . . woman with her legs in the air, standing on her head. 6. Leo, the lion—his nose and head. (Couldn't see the bat or butterfly). 2′28″
Card VI:	16″	7. Whole thing looks like a tree decoration—Christmas tree decoration . . . clown up at top of it. 8. Looks like a rug . . . a bear skin (. . . legs sticking out . . . soft . . . drawn) 2′38″
Card VII:	72″	9. A potato race. (Roll potatoes . . . those all look like potatoes). 3′40″
Card VIII:	36″	10. Some animal (the head, nose, mouth, eyes)
		11. An umbrella—just my imagination. 2′53″
Card IX:	29″	12. Looks like a Christmas tree decoration (all of it, the shape). 13. Looks like a mouse (nose, eye). 2′17″
Card X:	63″	14. Looks like a crab, the legs, body. It's squatting low and round. That's all. 3′01″

Summary

R12 T30′ 57″

W 2	M 1	H 1	F+ %	64	
D 12	F+ 7	A 7	A %	57	
Dd 0	F− 4	Ad 1	S	0	
	F 2	My 1	P	2	
	14	Re 1	T/1R	58″	
		Rl 2	T/R	138″	
Ap: W D. (Dd)		Jm 1			
Sequence		14	Exp. Bal. 1/0		

The Rorschach responses reflect the paucity of associations, perseveration and stereotypy, concreteness of thinking, and delayed reaction times characteristic of the brain-damaged patient. The breadth of associations in such an otherwise meager record, the sexual response in Card V, and the playful, recreational character of "tree decoration" and "potato race" suggest premorbid interests. The lowered function in the projective perceptual task of the Rorschach situation is consistent with the decrease in psychological functioning seen also in the more structured Wechsler tests.

SUMMARY

Early in the 18th century, Francis Joseph Gall reported the case of a man in whom the right hemisphere had been destroyed by disease but whose "intellectual faculties remained intact up to the time of death." However, it was not until Dandy's first hemispherectomy, in 1923, that operative procedures were carried out on such an extensive area in man. Since this pioneer operation there have been published reports on only 41 hemispherectomies, 28 done in this country, including Dandy's early series of 7 cases. With few exceptions, the preoperative and postoperative psychological functioning of the patients has been described only in general terms, indicating the need for a systematic follow-up study with standard, objective psychological measures.

In the present study, a man aged 54 had been operated on for an extensive brain tumor, and the entire right cerebral hemisphere was removed. Psychological

examination could not be begun until the 18th postoperative day, because of the sequelae of the radical operation. On nine occasions during five months following operation, the patient was examined with the Wechsler Memory Scale and various subtests of the Wechsler-Bellevue Intelligence Scale. Three months after operation, the Rorschach test also was administered. The examination data reflect wide variations in functioning, with concreteness and perseveration of ideas, confused and psychotic-like thinking, clang associations, mingling of old and new information, and self-reference. Also seen are premorbid levels of superior vocabulary and verbal facility and extremely compulsive behavior.

A number of reports on postoperative psychological functionings have emphasized the absence of disturbance following hemispherectomy, the generalization being made from clinical observations. The present study by means of objective psychological measures reflects extreme variation and disturbance in the psychological functioning after operation. A similar systematic follow-up study in a number of cases is suggested in order that the course of recovery of psychological function after hemispherectomy may be portrayed more accurately.

FRACTURES OF THE SPINE DURING INSULIN SHOCK THERAPY

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INTRODUCTION

IN A PREVIOUS communication,[1] an analysis of electric-shock fractures was presented in which correlations with possible predisposing factors were discussed.

It was the purpose of the present, similar invesigation to study insulin-shock fractures of the thoracic portion of the spine in an effort to correlate such fractures with any determinable theoretical predisposing or etiologic factors and to determine the incidence of such fractures with insulin shock treatment. A review of the literature did not reveal any information along these lines.

General Comment Regarding Insulin Shock.—Insulin shock is defined as a "circulatory insufficiency resulting from overdosage with insulin which causes the sudden reduction of blood sugar. It is marked by tremor, sweating, vertigo, diplopia, convulsions, and collapse." [2] Insulin shock therapy for certain psychoses was developed in 1928 by Manfred Sakel,[3] and the fact that it is still being used despite the difficulty of its administration is evidence in favor of its value as a therapeutic procedure. At the Brooklyn State Hospital,[3] for example, there were 1,128 insulin-treated patients and 876 control patients with schizophrenia. It was computed that insulin shock treatment saved 286,695 hospital-patient days and $80,274 in costs of food and clothing alone. At the Pennsylvania Hospital, it was estimated that improvement resulted in 37 to 48.8% of patients over a five-year period, as against 16% for control patients without insulin or other shock treatment.

In our present series, it would appear that similar results were obtained and that the treatment has definite merit despite its rigorous aspects and moderate complexity.

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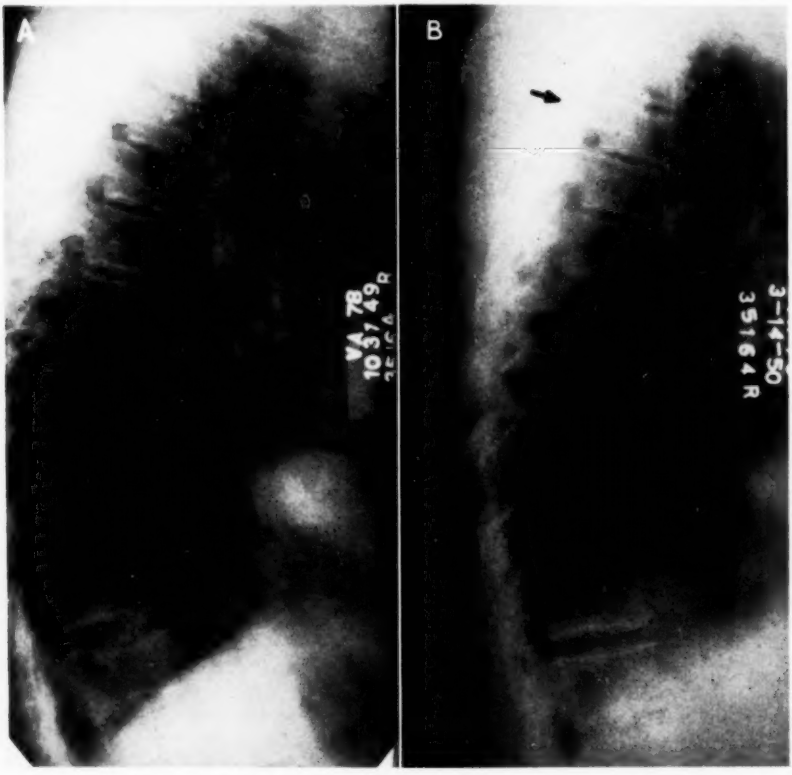
Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Meschan, I.; Scruggs, J. B., and Calhoun, J. D.: Convulsive Fractures of the Dorsal Spine Following Electric-Shock Therapy, Radiology **54**:180-193, 1950.

2. Dorland, W. A. N.: The American Illustrated Medical Dictionary, Ed. 20, Philadelphia, W. B. Saunders Company, 1944.

3. Shurley, J. T., and Bond, E. D.: Insulin Shock Therapy in Schizophrenia, Veterans Administration Tech. Bull. TB 10-501, 1948.

At this Veterans Administration Hospital, the original method, as advocated by Sakel, by which the dose of insulin is increased day by day from 10 to 20 or 30 units, as well as the method presented in the Veterans Administration Technical Bulletin TB 10-501, by Shurley and Bond,[3] is used in an attempt to bring about the desired stage of coma.



Fractures of the thoracic portion of the spine in insulin shock in a white man aged 40, 62 in. (157.5 cm.) in height and 120 lb. (54.4 kg.) in weight, with a diagnosis of schizophrenia, paranoid type. (*A*) the control film, on Oct. 31, 1949, showed slight deforming spondylosis localized to the lower thoracic and lumbar portions of the spine, but the upper thoracic portion of the spine gave no manifestations of abnormality. Insulin therapy was started on Feb. 15, 1950, and hypoglycemia (Stage 1) was achieved on February 19. A dose of 140 units of insulin was required at this time. There were 20 comas thereafter, with an average duration of each coma of approximately 45 minutes. No convulsions were recorded at any time in this patient's history. The restraints employed during insulin coma were sheet ties, and he had no relapses of coma.

(*B*) A film taken after this series of insulin treatments, on March 14, 1950, showed superior-end-plate sclerosis and compression of several of the upper thoracic vertebral bodies, particularly of the third and fourth. He achieved slight clinical improvement, and after this course of therapy it was considered that he could have partial privileges in the open ward. It is noted that the superior-end-plate indentation and the sclerosis of superior end-plates of the third and fourth thoracic vertebrae is rather the usual finding not only after insulin shock but after electric shock. This case is noteworthy, however, particularly in that no convulsions were recorded during the patient's series of treatments.

The hypoglycemia produces profound changes in the entire body, but especially in the central nervous system, since glucose is the sole metabolic fuel of the tissues of the central nervous system.

The lowest and oldest parts of the central nervous system have the lowest rates of oxygen consumption; the highest centers, the highest rates. Therefore, the highest centers suffer first from hypoglycemia, and the lowest, last. Thus, one can divide the stages of hypoglycemia (after Himwich) into five stages of hypoglycemic coma:

1. Cerebral and cerebellar depression: perspiration, salivation, muscular relaxation, fine tremor, somnolence, clouded consciousness, excitement.

2. Subcortical-diencephalic release: release of subcortical motor nuclei, producing loss of environmental contact, forced grasping, primitive movements, myoclonic twitchings, clonic spasms, and motor restlessness; thalamic release, producing sensory changes and increased sensitivity to stimulation; hypothalamic release, producing changes in the autonomic nervous system, with increase in sympathetic activity.

3. Release of the midbrain, producing diminished sensitivity, tonic spasms, independent movements of the eyes, Babinski reflex.

4. Release of the medulla, producing extensor spasm.

5. Increased parasympathetic activity, muscular flaccidity, depressed reflexes, and loss of the corneal reflex.

Insulin Convulsion.—Two types of convulsions may occur in hypoglycemia: an "early" type, which occurs typically in the second stage of hypoglycemia, is almost entirely safe, and requires only the prevention of tongue bite and the interruption with glucose; and a "late" type, which occurs typically in the fourth or fifth stage of hypoglycemia, is always dangerous, and demands immediate intravenous termination with dextrose. Either complication, successfully managed, usually proves helpful to clinical improvement.

A convulsion is always an indication for the immediate interruption of treatment for that day, and rest periods of one to several days are inserted after the convulsion or any reaction considered serious by the therapist.

Ordinarily, about one-half of all patients will have at least one spontaneous convulsion during their course of treatment. It usually occurs in the first or second week of their treatment, in the second stage of insulin hypoglycemia, if the latter is prolonged more than one hour. However, it may occur at any time after the treatment is started. Occasionally, it may occur after consciousness has been regained.

Combined electroconvulsive and hypoglycemic therapy is used in about one-third of the cases. At this hospital there are four reasons for the combined use of electric- and insulin-shock therapy: (1) when there is profound affectivity coloring; (2) when there is extreme overactivity or uncooperativeness; (3) when, in the later stages of insulin treatment, it is desired to stabilize or fix the improvement brought about by the insulin therapy; (4) when insulin shock has produced less improvement than was desired. Depending on the affective coloring, the overactivity, and other factors, electric shock was given an irregular number of times to patients undergoing

insulin-shock therapy, and frequently irregularly throughout the course of treatment. In general, electric shock was given to these patients on alternate days, three times a week, at the time that the patient was in Stage-2 or early Stage-3 coma.

METHOD OF STUDY

A series of 166 cases was available with all the data complete. This number was obtained from 187 consecutive cases, in which insulin shock therapy was given at the Veterans Administration Hospital, North Little Rock, between Jan. 1, 1947, and July 15, 1950, inclusive. Twenty-one cases were omitted because all the data were not available.

A lateral roentgenogram of the thoracic portion of the spine was part of the routine work-up before insulin shock therapy was begun, and a second roentgenogram was obtained at the completion of the series. Some of the patients had roentgenograms during their course of therapy, especially the ones who sustained a fracture or who manifested clinical symptoms suggesting fracture.

There were four groups of patients, depending on whether they had had previous or immediate therapy:

Group 1: The patients received insulin shock therapy alone and had no history of previous shock therapy of any type or x-ray evidence of spinal fractures. Forty-six of the 166 patients were in this category.

Group 2: The patients received insulin shock therapy some time after electric shock or metrazol® (pentylenetetrazole U. S. P.) shock during a previous hospitalization elsewhere. The insulin shock, however was given without any concomitant shock treatment. A total of 53 of the 166 patients comprised this group.

Group 3: The patients received insulin shock combined with electric shock while they were in insulin coma, as described above. These patients had had no previous shock therapy. Thirty-two of the 166 patients were in this group.

Group 4: The patients received insulin shock therapy combined with electric shock while they were in coma. Unlike Group 3, these patients had received other forms of shock therapy prior to the present treatment. Thirty-five of the 166 patients were in this group.

The factors correlated with the insulin-shock fractures were age, sex (all the patients were males), race, weight, clinical diagnosis, number and duration of grand mal seizures resulting from the insulin shock and/or electric shock therapy, number of days insulin was given before the desired stage of hypoglycemia was achieved, amount of insulin required to induce coma on the day it was obtained, total number of comas, average duration of each coma, type of restraint during coma, and any appearances on the control films of a condition which might predispose to fracture.

Preliminary observations were also recorded relating to clinical improvement following treatment.

GENERAL OBSERVATIONS

In Group 1, consisting of persons for whom insulin shock therapy was the only form of treatment employed, six of the 46, or 13%, sustained a spinal fracture. This was the lowest incidence of fracture in the four groups; and, indeed, it is considerably lower than the 33%[1] incidence of fractures which has been reported for electric shock alone (or metrazol® shock alone, elsewhere).

The incidence of fractures of the spine in Group 2, consisting of patients who had had electric shock prior to the present insulin series was 10 out of 53, or 19%. These patients all had "control" roentgenograms prior to induction of the insulin shock, and hence all the fractures seen were attributed to the insulin shock therapy alone.

In Group 3 there was no previous history of shock therapy, but in this group insulin shock and electric shock treatments were combined. Eight patients out of 32, or 25%, sustained fractures of the spine.

In Group 4, in which there was a history of previous shock therapy, as well as the combined use of insulin and electric shock at the time of the study, the incidence of spinal fractures was 6 out of 35, or 17%. Since these patients had usually been found to be resistant to spinal fracture in their previous shock treatments, it is understandable that the incidence of fracture in this group would be lower than in a fresh, untreated series of patients. The number of patients in each group is relatively small, and extensive deductions are not permissible.

The over-all incidence of spinal fracture in all 166 patients was 31, or 18%, as compared with 33% for electric shock (and for metrazol* shock from series reported elsewhere).

Age and weight had no apparent bearing on the incidence of fractures in any of the groups; and since practically all of the patients were white men, no investigation of the correlation with sex or race was possible.

Correlation with Number of Days Insulin Had to be Administered Before Any Stage of Coma Was Achieved.—In group 1, 29 of the 46 patients experienced coma within 10 days, and 37, within 15 days; 2 patients required 16 to 20 days; 2 required 46 to 50 days, and 5 never reached Stage 3 of hypoglycemia. All the fractures were sustained by patients requiring 15 days or less of insulin to experience some stage of coma.

In Group 2, the findings were very similar, with 50 of the 53 patients experiencing coma in 15 days or less and all of the 10 fractures occurring in these 50 patients. Of the other three patients, two required 16 to 20 days to achieve coma, and one never achieved a Stage-3 coma.

In Group 3 the findings were also very similar, with 31 of the 32 patients experiencing coma in 15 days or less and all of the eight cases of fracture occurring in this group. One patient required 20 days to achieve coma.

In Group 4, the findings were similar with one exception: A spinal fracture occurred in a patient requiring 23 days to experience coma, whereas, all the other spinal fractures occurred in those 28 patients (out of 35) who required 10 days or less to experience some stage of coma. In this group, in addition, one patient was in the group requiring 16 to 20 days to achieve coma; two were in the group requiring 26 to 30 days, and one never achieved a Stage-3 coma.

Of the entire group of 166 patients, 125 (75%) experienced some stage of coma in 10 days or less, and of the total of 31 fractures, 24 (77%) occurred among these 125 patients. Twenty-three patients (14%) required 11 to 15 days to experience coma, and of these, 6 (26%) sustained spinal fractures.

It is probable, therefore, that the number of days required to achieve Stage-3 coma has some correlation with the occurrence of spinal fractures, and there was a tendency for insulin-resistant patients not to sustain fractures.

All four groups were quite similar in this regard.

It should be noted that in every instance the x-ray studies were obtained after the completion of the treatment series, and hence the exact time of occurrence of the fractures in the sequence of comas is not known:

Correlation with Amount of Insulin Required to Induce Coma per Day.—Seven patients did not experience a Stage-3 coma even with 800 or more units of insulin, and five of these were in Group 1.

In Group 1, 33 of the 46 patients (72%) required 300 units or less to achieve some stage of coma, and the six fractures occurred in patients utilizing 200 units or less. Three of these fractures occurred with convulsions and three without.

In Group 2, 45 of the 53 patients (85%) experienced some stage of coma with 300 units or less, and 8 of the 10 spinal fractures occurred among these, the remaining 2 patients with fracture requiring 400 and 600 units, respectively. (One patient requiring 300 units sustained a fractured humerus, but no fracture of the spine.)

In group 3, 30 of the 32 patients (90%) required 300 units or less to experience some stage of coma, and all eight of the spinal fractures occurred among these 30 patients.

In Group 4, 30 of the 35 patients (86%) required 300 units or less to experience some stage of coma, and all six fractures were within this group.

In the entire group of 166 patients, 125 (75%) experienced coma beyond the second stage of hypoglycemia with 200 units of insulin per day or less, and 26 (84%) of the 31 fractures occurred among these. Thirteen patients required 200 to 300 units, and of these, three sustained fractures. All but two (3%) of the fractures were sustained with 300 units of insulin or less, although 28 of the 166 patients (17%) required more than that amount of insulin to experience coma, or they never experienced at least a Stage-3 coma.

Thus, it would seem that fractures were more likely to occur in patients who required 200 units of insulin or less to experience some stage of coma, and again it would appear that insulin-resistant patients were less likely to suffer fractures of a thoracic vertebral body in insulin shock therapy. Fractures in Groups 1 and 2 (in which no supplementary electric shock was given during coma) occurred just about as frequently in patients without insulin convulsions as in those with insulin convulsions.

Correlation of the Number of Stage-3 Comas [4] to Incidence of Spinal Fracture with Insulin Shock.—Usually 50 comas were considered maximum treatment for any patient; however, in Group 2, one patient had 55 Stage-3 comas, and another had 65; in Group 3, one patient had 70 Stage-3 comas, and in Group 4, five patients had 51 to 60 such comas.

In Group 1, 32 of the 46 patients (70%) had 16 to 35 comas, and all the six spinal fractures occurred in these.

In Group 2, 31 of the 53 patients (60%) had 16 to 35 comas, and only 5 of the 11 fractures occurred in this group (46%). The fractures were distributed irregularly, quite independently of the number of comas.

In Group 3, 9 of the 32 patients (28%) were given 36 to 40 comas; otherwise the distribution was rather an equal one. Two of the eight fractures (25%) occurred in this group, and here, too, the fractures were very evenly distributed among the groups receiving 16 to 50 comas.

In Group 4, most patients received between 16 and 60 comas, and there was no special predilection for a given number of comas. Likewise, there was no predilection for fracture to occur among patients with a given number of comas.

4. In the following pages, unless otherwise specified, the term "coma" refers to stage-3 hypoglycemia.

Of the total 166 patients, only 27 patients (16%) received less than 16 comas, and the number of comas were very evenly distributed, up to 50 comas. No close correlation could be drawn between the number of comas and the incidence of fracture.

Correlation of Average Duration of Each Coma to Spinal Fractures During Insulin Shock.—In group 1, 37 of the 46 patients (80%) had comas lasting from 16 to 90 minutes, and all the fractures occurred among these.

In Group 2, 43 of the 53 patients (81%) had comas lasting from 16 to 90 minutes, and all the spinal fractures occurred among these 43 patients.

In Groups 1 and 2, 17 of the 99 patients (17%) had comas lasting from 31 to 45 minutes and 7 of these (41%) sustained spinal fractures. The other fractures were without predilection for any duration of coma. The numbers involved were rather small, and hence conclusions are of questionable significance.

In Groups 3 and 4, the duration of coma was very evenly distributed, and the fractures were distributed likewise, so that no correlation with duration of coma can be drawn. Moreover, the superimposition of electric shock would introduce an additional factor, which would make the actual duration of the coma difficult to evaluate in the latter two groups.

This factor of duration of coma is, therefore, of doubtful significance. It is not likely that it predisposes to fracture of the thoracic portion of the spine.

Correlation of Number of Grand Mal Seizures with Spinal Fracture.—Of the 46 patients in Group 1, 30 did not have any grand mal seizures; 13 had one seizure; 2 had two seizures, and 1 had three seizures. Six fractures were sustained in this group—three among the 30 patients without grand mal convulsions, and three among the 16 with grand mal convulsions. It is indeed interesting and significant that fractures were sustained in the absence of recorded visible convulsions, despite extremely close observation by the nursing and medical staff.

A similar statistical observation was made in Group 2. Of the 53 patients in this group, 10 sustained fractures, 5 without any convulsive seizures and 5 with one convulsion.

In Groups 3 and 4 therapy was complicated by electric shock during coma, and it is perhaps significant that of the 67 patients in these two groups, fractures occurred in 14 (21%), with an incidence of 25% for Group 3 and 17% for Group 4. As is true of electric shock alone, practically all the fractures were sustained (except one in Group 4) within the first five seizures. Patients in Group 3 were similar to patients treated by electric shock alone, and the difference between 25 and 33% (for electric shock therapy alone) is not considered significant in view of the small number of cases (32) in Group 3. It is probable, however, that patients who have had previous electric shock therapy without fracture are less susceptible to fracture from subsequent convulsive therapy.

Clinical Improvement.—In this study, no effort is made to evaluate the neuropsychiatric aspects of insulin shock therapy. An early gross evaluation of improvement is readily made, however, by the following classification:

Marked Improvement: Patient was allowed to return home and remained fairly well adjusted.

Good Improvement: Patient remained institutionalized, but could carry open-ward privileges.

Fair Improvement: Only minimal improvement noted.

Temporary Improvement: Improvement of any type which did not last longer than three months.

No improvement.

According to this classification, in Group 1 (46 patients) three were markedly improved and nine were well improved—a total of 12 (26%) of the 46 patients. None of these 12 patients sustained fractures. Four had had convulsive seizures, and eight had not.

In Group 2 (53 patients) 3 experienced marked improvement, and 5, good improvement (15%). Four of these eight patients sustained fractures, and four did not. Five of the eight had had convulsions and three did not.

In Groups 3 and 4 (67 patients), 2 had marked improvement, and 10, good improvement (18%). Only 1 of these 12 patients sustained a fracture. They all had had convulsive seizures.

Incidence of Fracture of the Thoracic Portion of the Spine

	Group 1		Group 2		Group 3		Group 4	
A. Patients with Normal Control Films								
Convulsions	Without	With	Without	With	Without	With	Without	With
Incidence	1 in 20	2 in 12	2 in 12	2 in 11	0	3 in 19	0	2 in 15
Percentage	5%	17%	17%	18%	0	16%	0	13%
B. Patients with Previous Indentations or Narrowness of Vertebral Bodies on Control Films								
Convulsions	Without	With	Without	With	Without	With	Without	With
Incidence	3 in 8	0	4 in 15	1 in 23	0	7 in 13	0	4 in 15
Percentage	37.5%	0	26.6%	4.3%	0	54%	0	26.6%

The greatest improvement (26%) was therefore sustained in that group having only the insulin shock therapy, and no previous or simultaneous shock therapy. Moreover, convulsive seizures were not necessary to achieve this improvement.

There was no improvement, or only temporary improvement, in 18 of the 46 patients in Group 1 (39%); in 32 of the 53 patients in Group 2 (60%); in 16 of the 32 patients in Group 3 (50%), and in 15 of the 35 patients in Group 4 (43%).

In 81 (49%) of the total of 166 patients there was, therefore, no improvement; and 32 (19%) of the total number showed good and marked improvement. The rest (32%) fell into the category of fair improvement, without special indication from any special group.

Correlation of Spinal Fracture with Roentgenographic Findings on Control Films.—In view of the previous demonstration [1] of high correlation of the appearance of anterior narrowness of a vertebral body on the control film and the incidence of postshock spinal fracture, a special study was made of this factor. This correlation is indicated in the two parts (A and B) of the accompanying table. From this table, it is seen that in Group 1, whereas the incidence of fracture of the spine was 3 out of 32 patients with normal spines, it was 3 out of 8 patients who had spines with one or more vertebral bodies that were narrowed anteriorly or indented in their superior end-plates. These fractures occurred with or without recorded convulsions, and the site of the fractures was usually the fourth, fifth, or sixth thoracic vertebra.

There was no predisposition to fracture of the spine in kyphosis, minimal deforming spondylosis, or osteochondrosis, conditions which were present in approximately 15% of all patients.

In Group 2, 17.5% of the 23 patients with normal spines prior to insulin shock sustained fractures. Thirty-eight of this group had some evidence of injury to one or more vertebral bodies from their previous electric shock therapy. Only 5 of these (13%) sustained additional fractures. Thus, previous electric-shock fractures do not predispose to insulin-shock fractures, whereas anterior narrowness from other causes prior either to electric shock or to insulin shock does predispose to spinal fracture, although the incidence of fracture following insulin shock is less than one-half that following electric shock or metrazol® shock.

Again, there was no predisposition to fracture from kyphosis, minimal deforming spondylosis, or osteochondrosis.

In Groups 3 and 4, in which electric shock was combined with insulin shock, the incidence of fracture was 5 of 34 patients with normal spines (14.7%), but when there was anterior narrowness of a vertebral body prior to present shock it was 11 of 28 patients (40%). Among the latter, the incidence of fracture was 54% in Group 3 and 27% in Group 4 (but the numbers in each group are too small for significant comparison).

No other predisposing factor was demonstrated.

It is interesting to note that the incidence of fracture among patients with normal spines was virtually the same in all four groups, but was significantly higher in patients whose spines prior to shock therapy had some anterior narrowness of the vertebral bodies. In the latter group, the highest incidence of fracture was obtained when insulin shock and electric shock were combined in a patient who had not had previous shock therapy.

EXPLANATION OF OCCURRENCE OF SPINAL FRACTURE WITH INSULIN COMA

As for the manner in which spinal fracture occurs, it is our belief that the mechanism must be similar for all types of convulsive therapy.[1] The cervical portion of the spine and the skull are pulled caudad by the longer upper-back muscles, and these structures deliver a "hammer blow" to the superior end-plates of the upper thoracic vertebrae, which at first curve away from the cervical portion of the spine. This blow gives rise to the superior end-plate impression on the third, fourth, and fifth thoracic vertebrae, which is the earliest manifestation of injury. Subsequently, or simultaneously, the smaller, but powerful, flexor muscles of the spine may superimpose the element of anterior compression.

It apparently requires only a few convulsions to produce this injury, and hence it is probable that even the "early" insulin convulsions may account for some of these injuries. Although no convulsions are recorded for many of the insulin-treated patients who sustain fractures, it is probable that many "early" convulsions, particularly, may not have been recorded, since they represented a Stage-2 (and not a Stage-4 or Stage-5) phenomenon. Perhaps even some of the muscular twitchings and clonic spasms of Stage 2, or the torsion and tonic spasms of Stage 3, may be effective in this regard; and yet these phenomena would hardly be classified and recorded as convulsions.

PREDISPOSING FACTORS

The only significant predisposing factor to fracture which has consistently appeared in both our electric-shock and our insulin-shock studies has been the appearance of anterior narrowness in one or more of the vertebral bodies prior to therapy. Thus, in the present series, 89 of the patients had thoracic vertebrae considered to be normal roentgenographically, and of these, 12 representing all four groups, sustained fractures (13.5%). There were 74 patients with narrowness of one or more vertebral bodies on the control film; of these, 19 (25.6%) sustained compression injuries of the spine. The same ratio of approximately twice as many fractures in patients with this spinal defect was demonstrated previously for electric shock alone also.[1]

It is not necessarily the anteriorly narrowed vertebral body which undergoes compression, and indeed it usually is not. It is our belief that the anterior narrowness in any of the vertebral bodies, when unaccompanied with previous trauma or shock therapy, may indicate an inherent defect in ossification or a developmental deficiency—and hence other vertebral bodies most subject to strain under these circumstances of shock become fractured by the shock therapy.

The complication of spinal injury is slightly less than one-half as frequent with insulin shock as with electric shock. When one combines electric and insulin shock, spinal fractures occur in highest incidence in those patients with anterior narrowness of vertebral bodies on a control film—and in this group particularly curare carefully administered alone with the electric shock could probably diminish the hazard of the fractures.[1]

Those patients who were "insulin-resistant" and required many days or high doses of insulin to achieve a desired coma did not seem quite so likely to sustain spinal fractures.

Improvement (short period of observation) was good or marked in about one-fourth of the patients receiving shock treatment for the first time in the form of insulin coma alone (Group 1), in about one-fifth of those who received shock treatment for the first time in the form of insulin coma combined with electric shock (Group 3), and in about one-sixth of those who had had electric shock previously and had insulin coma either alone or combined with electric shock (Group 2 and 4). These evaluations are "short-term" gross ones only.

SUMMARY

The thoracic portions of the spines of 166 psychotic patients (all men) treated with insulin shock, 99 without and 67 with combined electric shock, were studied. Of these 166 patients, 78 had had no previous shock therapy, and 88 had had previous electric shock treatments. Of the 78 who had had no previous shock therapy, 46 were treated with insulin shock alone, and 32 with electric shock superimposed on insulin coma.

The findings for each group of patients were analyzed from the standpoint of spinal fracture.

1. It was found that the over-all incidence was 18%—approximately one-half the incidence of spinal fracture with electric shock (or metrazol® shock in studies elsewhere).

2. As was the case with electric shock, the incidence of spinal fracture was twice as great if on the films prior to the treatment any anterior narrowing of vertebral bodies was noted.

3. Fractures occurred less frequently in insulin-resistant patients.

4. Spinal fractures occurred with approximately equal frequency in patients with and in patients without recorded grand mal convulsions during insulin coma. A possible explanation of this phenomenon is presented.

An early evaluation of clinical improvement achieved is presented so that a gross evaluation of the relative importance of the complication of fracture may be attained. Roughly, improvement was good or marked in one of four patients treated for the first time with insulin shock alone, in one of five patients treated for the first time with combined insulin coma and electric shock, and in one of six patients who had had previous treatment and were now treated by either insulin shock alone or insulin shock combined with electric shock. In view of the relatively small numbers in each group, no far-reaching conclusions have been drawn in regard to the clinical value of insulin shock therapy.

The research committee of the Veterans Administration Hospital, North Little Rock, Ark., made a critical analysis of the psychiatric data in this paper. Dr. Charles R. Rayburn placed at our disposal the insulin work sheets on many of these patients and made a critieal analysis of the results presented here.

PHENOMENON OF REDUPLICATION

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IN STUDIES of the behavior of patients with organic brain disease the phenomena of reduplication for place, person, and time have been observed. Reduplication for place may be defined as the confabulation of the existence of two or more places with almost identical attributes, although only one exists in reality. Reduplication for person is the confabulation that there are two or more persons with almost identical attributes, although only one exists in reality. Reduplication for time is the confabulation that a present experience has also been experienced at some time in the past.

In 1903 Pick [1] described reduplication for place and person and termed it "reduplicative paramnesia." As an example, he cited the case of a patient with a diagnosis of senile dementia who, while in Pick's clinic in Prague, said that she had been in another clinic in another city, although the two clinics were exactly alike and a professor of the same name headed each clinic. Without referring to Pick's concept, Henry Head [2] described a similar case. A soldier with a bullet wound in the frontal region "thought that there were two towns of Boulogne, one of which, on the homeward journey from the front, lay near Newcastle; the other one, in France, was reached after you had crossed the sea." Head stated that the man appeared to be rational in all other respects, except that he wrote letters to his mother while recognizing the fact that she had been dead for many years. Recently there has been renewed interest in reduplicative phenomena, and instances of reduplication for place have been reported by Paterson and Zangwill [3]; Bender, Furlow, and Teuber,[4] and Weinstein and Kahn.[5] In the present paper, it is pointed out that reduplication is not a neurological rarity, but a significant pattern of behavior.

This study was aided by the Neurological Research Fund.

From the Neurology Service of the Mount Sinai Hospital.

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CASE MATERIAL

Reduplicative phenomena were observed in 16 patients with organic brain disease. Two patients had meningoencephalitis. Three patients had had subarachnoid hemorrhages, with aneurysms of the circle of Willis, demonstrable in arteriograms. Four patients had tumors in the region of the third ventricle, demonstrable in ventriculograms, and in three instances verified at operation. Three patients had intracranial metastases. One patient had a cerebellar abscess. One patient had an acoustic neurinoma with increased intracranial pressure. One patient had a deep-seated hemorrhage into the right cerebral hemisphere. In one patient reduplication developed during a course of electric shock treatment for intractable pain associated with a neoplasm of the lower thoracic portion of the spinal cord. In one patient a clinical diagnosis was never made with certainty. The electroencephalograms of all 16 patients showed diffuse delta activity, the frequency of the waves ranging from 1 to 6 cps.

OBSERVATIONS

Reduplication for Place.—This phenomenon was observed in 16 patients. Patients exhibiting this behavior stated that there were two, sometimes more, hospitals bearing the same name and having similar characteristics in varying degree. Frequently the personnel were described as the same. The duplicated institutions were differentiated in size and location, in terms of "better" or "worse," in the severity of the cases handled, and by some important event in the patient's experience, such as an operation, which was said to have occurred in one place but not in the other. The place in which the patient claimed to be at the time of questioning possessed features that seemed to solve his personal problems, mainly those relating to his illness. Frequently the patient characterized the hospital in which he was as a smaller branch of the main one and located it closer to his home. One man said that he was in the Mount Sinai Hospital in Miami Beach, which was a convalescent place, but that he had previously been in the Mount Sinai Hospital in New York.

A woman manifested duplicative disorientation during a course of electric shock therapy, given for the intractable pain associated with a neoplasm of the spinal cord. After having been irritable and complaining of severe pain, she became euphoric and said that she felt fine. At this time she said that the "old Mount Sinai Hospital was terrible" and the nurses had been cruel, but that although nurses in the "new Mount Sinai Hospital" were the same, they were very kind and considerate and the hospital was "wonderful." A man, after a craniotomy for brain abscess, said he had been operated upon in the first hospital but that in this one they did not take cases of such a serious nature. Various degrees of partial duplication were also seen in which the hospital in which the patient claimed to have been previously bore a related name, such as "Mount Holyoke" or "Mount Simonai."

Reduplication for Person.—This phenomenon was seen in five patients and was always associated with reduplication for place. One patient claimed that his nurse had a daughter who also took care of him. He said they looked exactly alike except that the "mother" wore glasses. Another patient with one living and one dead sister confabulated that she had two living sisters. The living sister's name was Margaret, and she was also called Maggie. The patient, however, said that she had two living sisters, one named Margaret and one named Maggie.

Reduplication for Time.—This phenomenon was observed in 11 patients. It was common in the initial interview for the patient to state that he had met the

examiner previously, confabulating that he was an acquaintance or that he had business dealings with him. Frequently this kind of temporal reduplication persisted for weeks after other manifestations of abnormal behavior had disappeared. A girl recovering from meningoencephalitis repeatedly remarked that the examiner had been a professor at her college. She also expressed the feeling that she had previously known all the patients in the ward. A man with subarachnoid hemorrhage and left hemiplegia, which he denied, insisted that the patient in the bed across from him had worked in his garage. As in the other instances of reduplication, these feelings were of great intensity and would endure in the face of all logical arguments. One woman thought that the examiner was an insurance salesman who lived in her home town. She consistently greeted him as "Mac." One day she finally seemed to accept the explanations of her daughter and the doctor that he was not "Mac," but only bore a slight resemblance to him. After this she chatted for several minutes, then remarked, "Well, Doctor, if this business doesn't turn out too well, you can always go back into insurance."

OTHER ALTERATIONS IN BEHAVIOR

Reduplicative phenomena were never seen as solitary manifestations of abnormal behavior, although, as mentioned, they might outlast the other aspects. Patients with reduplication manifested it in more than one sphere. At some time in the course of the illness the patients also showed such alterations of behavior as other patterns of disorientation for time and place, other confabulations, paraphasic language, and mood changes, mainly of a euphoric or a paranoid type. Twelve of the 16 patients denied their illness or defects (anosognosia).

REPORT OF A CASE

The following case is illustrative of reduplication for place and person.

M. S., a woman aged 53, was admitted to the Mount Sinai Hospital on March 22, 1950. For the preceding three months she had complained of weakness, abdominal pain, and loss of weight. Hoarseness, headaches, and dizziness had been present for three weeks, and nausea and vomiting for one week. The results of the general physical examination were unremarkable. A roentgenogram of the chest showed an area of density in the right lower lobe, interpreted as a primary neoplasm. Under observation, the patient became apathetic, drowsy, and incontinent of urine. Neurological examination on April 8, 1950, showed bilateral papilledema and awkwardness of the right upper extremity. She was disoriented for time and place, giving the date as January, 1945, and stating that she was home. An electroencephalographic record showed symmetrical and diffuse 4 to 6 cps delta-wave activity with superimposed 2 to 3 cps bursts. A ventriculogram revealed symmetrical dilatation of the lateral ventricles and the anterior part of the third ventricle. On April 13 a suboccipital craniotomy was performed by Dr. Sidney Gross, and a metastatic neoplasm, 1 in. (2.5 cm.) in diameter, was removed from the right cerebellar hemisphere. The patient recovered well from the operation and was seen by us daily until her sudden death, from respiratory obstruction, on May 19.

During the week following the operation the patient was restless, drowsy, and incontinent of urine. She denied that she was ill or had had an operation. She was disoriented for time and place, saying that she was home and, later, in "Mount Morris Hospital." During the second postoperative week she seemed more alert, was cheerful, and still denied her operation. During the following week her sphincteric incontinence improved; she became oriented for time and aware of her operation. In the interview of April 28, she named and located the hospital correctly but showed reduplication for place, maintaining that there were two Mount Sinai Hospitals and that the hospital in which she was at the time was situated on F Place in the

Bronx, four blocks from her home. (Mount Sinai Hospital is actually located at 5th Avenue and 100th Street, in the borough of Manhattan. The patient lived in the neighboring borough of the Bronx, about 6 miles from the hospital).

The patient thought that the "two hospitals" were owned by the same people and stated that the one in which she was staying was a "sort of branch, although they don't charge you any less." She remained firm in her belief that the hospital was only four blocks from her home, remarking on several occasions, "I know I can walk it in a few minutes." She was shown a map of New York City and was asked to locate both her home and the hospital. She was able to do this correctly, but said, "I think it's four blocks, but on the map it's different." She recalled taking the subway to the hospital (at its correct address on 100th Street) for roentgenograms and going back by taxi, but said that the hospital in which she was at present was a smaller place, which did not take roentgenograms. Although able to identify correctly the park across the street from the hospital as Central Park and admitting that the park was in Manhattan, and not in the Bronx near her home, she closed the discussion by commenting that there must be some reason that her home and the hospital were so close.

This patient also manifested a reduplicative disorientation for person. She was actually the mother of 26-year-old twins, a boy, William, whom she usually called Bill, and a girl, Hilda. She maintained, however, that she had three children, twin sons, named Bill and "Willie," and a girl, who was "extra." She said that she had not seen "Willie" since the day before Christmas and that she was told he was recuperating from an illness. The patient was able to give detailed histories and descriptions of the "two sons." They had enlisted in the Army at the same time and each had been a sergeant "with a T" (her son's actual rank). On another occasion she said that "Willie" had been only a corporal. Bill had returned home on the Queen Mary, "Willie," on the Queen Elizabeth. Each was employed as a commercial artist (her son's actual job). They lived at home, sharing twin beds, and "Willie's" clothes were still at home hanging in the closet. They were inseparable but did not have the same friends. Bill had changed his surname the year previously (correct), but "Willie" had not. At the time "Willie" had said, "Let him go change his name—who cares?" Bill was taller, heavier, more athletic, and more popular with girls. Both boys resembled her, but she remarked that "Willie thought more like me."

The patient brought out a photograph of two young children, claiming that they were Bill and "Willie." When it was pointed out that one child was obviously a girl, the patient admitted that it was her daughter, but said, "Willie must have been running around somewhere; you know how hard it is to get children in a picture."

The reduplicative delusion for person was first noted in the patient a week after the operation. At that time she was voluble and spontaneous, and her family thought she was joking. Gradually she expressed more concern about the "vanished son," said he must really be very sick, and accused people of not telling her about him. On April 30 she asked, "What happened to Willie? Where is he? They told me six weeks ago he was recuperating. Something must have happened Christmas week, and I should know about it. He said he'd come back, but he never did." During the last three weeks of her life she no longer mentioned "Willie" spontaneously and became tearful and agitated when the subject was brought up. On May 4 she began to cry when asked about "Willie," insisting that she had three children and that "Willie" had been in an airplane accident. On May 9 she remarked, in an agitated fashion, that she "only had two children now." On another occasion she said, "I feel as if I've lost a son."

Reduplication for place and person persisted until the patient's death. During the last two weeks of her life the reduplicative delusions were the only abnormalities in her behavior that could be elicited. During this period she became more depressed and irritable and complained of increasing weakness. An electroencephalogram taken on May 10 showed only a small degree of diffuse and symmetrical 6 cps activity. On May 19 difficulty in breathing suddenly developed, and she died.

COMMENT

Pick[1] used the term reduplicative "paramnesia" because he attributed the phenomena to a defect in memory. This would not, however, explain why many patients with profound memory loss do not manifest reduplication, while patients

with reduplication may show little or no memory impairment (as in the case of M. S.). Pick's concept also fails to allow for the fact that all patients with reduplication, including Pick's own patient, manifest other abnormalities of behavior, such as disorientation, mood changes, and other delusions and confabulations. Finally, Pick did not take into account the extremely important factor of motivation in determining the elements of the reduplicative pattern. Thus, the hospital in which the patient "places" himself usually is the one that takes less serious cases or is closer to his home. In the case cited it was the "other" son who did not disappoint his mother by changing his name.

In considering the relation of the reduplicative delusions to the brain disease, it is difficult to conceive of reduplication as representing any unitary defect, such as loss of memory. The phenomenon, likewise, cannot be attributed simply to the effects of destruction of a particular area of the brain. In all cases, the pathologic condition was that which, by virtue of diffuseness, bilaterality or midline situation, or the presence of increased intracranial pressure or subarachnoid bleeding, produced diffuse delta-wave activity in the electroencephalogram. Thus, lesions in the diencephalon or midbrain are more likely to be associated with reduplication and the accompanying alterations in behavior than lesions limited to one hemisphere. We also observed a striking instance of temporal reduplication in a woman who had had a prefrontal lobotomy for relief of pain, but we have not included the case in the series because of insufficient data concerning the operation and the immediate postoperative behavior.

In studies of anosognosia,[5] disorientation,[6] and paraphasia[7] it was stated that these phenomena occur as aspects of a new integration of brain function in which there is an altered perceptual-symbolic system. Thus, patients identified objects in terms of only one aspect of their structure or function and were able to appreciate accurately only one of two simultaneously applied tactile stimuli. There was substitution of an experiential type of symbolism for the ordinary referential symbolism used by the normal adult in the waking state. Thus, the name of the hospital was no longer only a referential symbol serving purposes of identification and communication; it was also a means by which the patient might express his feelings. A paranoid patient called the hospital "Mount Cyanide"; a depressed patient called it "Misericordia." In reduplication, likewise, the person uses spatial, temporal, and personal modalities as vehicles for the expression of his needs and feelings. In the case cited, the patient, through the delusion of another son, was able to express her ambivalence toward, and identification with, her real son. It is significant that motivational factors determine the elements, but not the pattern, of reduplication. Two patients who did not deny their illness stated that the hospital in which they were at the time of the interview was larger and took the more serious cases. This contrasted with the statements of patients with anosognosia who reinforced their denial by asserting that the present hospital took only milder cases. In each instance reduplication occurs as an intrinsic pattern, but the elements, such

6. Weinstein, E. A., and Kahn, R. L.: Syndrome of Anosognosia, Arch. Neurol. & Psychiat. **64**:772-791, 1950.

7. Weinstein, E. A., and Kahn, R. L.: Nonaphasic Misnaming (Paraphasia) in Organic Brain Disease, A. M. A. Arch. Neurol. & Psychiat. **67**:72-79, 1952.

as type of case taken and size of hospital, varied with the particular motivation and personality. In the case described it was significant that the reduplicative delusions were most effective in "solving" the patient's problems during the period of the greatest alteration in function. In the first two weeks after her operation, when the electroencephalographic record was most abnormal, she was euphoric, denied her illness and operation, and talked freely of the "missing" son. During the last few weeks of her life, when her electroencephalogram was much less abnormal, she was depressed, anxious, and unwilling to discuss her delusion. Between the depressed and the euphoric stage there was a paranoid episode, in which the patient accused people of lying to her about the vanished "Willie." This pattern of progression has repeatedly been noted in patients with delusions, mainly those of denial of illness, and indicates the importance of levels of functional organization, as well as psychodynamic factors.

Under certain conditions reduplication may be present in persons without brain disease. The déjà vu phenomenon, which is comparable to temporal reduplication, may occur in convulsive seizures or in persons without any neurological abnormality. In discussing the feelings of reminiscence in patients with dreamy states, Jackson [8] used the term "mental diplopia" to denote the coterminous aspects in which a situation can be experienced. During World War II one of us (E. A. W.) was told repeatedly by soldiers of déjà vu phenomena occurring while they were approaching some dangerous situation. It was frequently accompanied by a euphoric feeling. This experience suggests a mechanism whereby a scene is duplicated and one of the parts projected into the past to a time, perhaps, when there was no danger. Reduplication may occur in dreams, the similarity of sleep to the functional state of the patients described having previously been pointed out.[9] Reduplicative phenomena also appear in the fantasies of children, such as the "good mother" and the "bad mother," or the imaginary companion who does the bad things. In addition to the difference between the child's electroencephalographic pattern and that of adults, Bender, Fink, and Green [10] have shown that in their reaction to simultaneous tactile stimuli children's perceptual organization is similar to that seen in patients with organic brain disease.

Reduplication does not appear to be a random manifestation of a confused state or a hysterical reaction following brain injury, but seems to be an orderly pattern of behavior integrated with brain function. The lesion does not create the phenomenon, which, in itself, may occur in the absence of demonstrable organic alteration. The effect of disease is to make it more enduring, more rigid, more stereotyped, and hence more easily recognized.

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8. Jackson, J. H.: Selected Writings of John Hughlings Jackson, edited by James Taylor and others, London, Hodder & Stoughton, Inc., 1932.

9. Jackson.[8] Cox, L. B.: Tumors of Base of Brain: Their Relation to Pathological Sleep and Other Changes in Conscious State, M. J. Australia **1**:742-752, 1937. Doty, E. J.: Diagnosis and Treatment of Delirious Reactions, M. Clin. North America **32**:647-654, 1948. Sandifer, P. H.: Anosognosia and Disorders in Body Scheme, Brain **69**:122-137, 1946. Wolff, H. G., and Curran, D.: Nature of Delirium and Allied States, Arch. Neurol. & Psychiat. **33**:1175-1215, 1935.

10. Bender, M. B.; Fink, M., and Green, M.: Patterns in Perception on Simultaneous Tests of Face and Hands, A. M. A. Arch. Neurol. & Psychiat. **66**:355-362, 1951.

SUMMARY

The phenomena of reduplication for place, person, and/or time in 16 patients with organic brain disease are defined and illustrated. One case is presented in detail.

The character of the pathologic process in the brain and the location of the lesions were varied. In all instances the electroencephalographic record showed bilateral diffuse delta activity.

Reduplication occurred as part of a general alteration in behavior, which included other types of disorientation for time and place, other confabulations, paraphasic language, and, in 12 cases, denial of illness or defect.

Reduplication was not explicable on the basis of a unitary defect but appeared to be a manifestation of a pattern of functional reorganization in the damaged brain. The elements of the pattern are in large part determined by motivational factors.

Reduplicative manifestations in persons without demonstrable organic brain disease are described as occurring in déjà vu phenomena and the fantasies of children. The relation of these phenomena to those found in cases of organic disease is discussed.

Dr. Sidney Gross permitted us to study the patient whose case history is reported.

N-BENZYL-β-CHLOROPROPIONAMIDE (HIBICON®)

A New Approach to Anticonvulsant Therapy

C. D. HAWKES, M.D.
MEMPHIS, TENN.

MODERN neurosurgical techniques have made possible the successful treatment of some patients with epilepsy by cortical excision or resection of the temporal lobe. At present, however, these methods are applicable to only a small percentage of the total number of patients with chronic convulsive disorders (Penfield and Erickson,[1] Penfield and Steelman,[2] Walker,[3] Morris[4]). We must, therefore, continue to treat most patients with chronic epilepsy with anticonvulsant drugs, supplemented by psychotherapy and occupational rehabilitation.

Anticonvulsant medication also has its limitations as a method of treatment. Walker[3] has estimated that anticonvulsant drugs will eliminate the attacks in about one-half the cases of chronic epilepsy and greatly decrease seizures in 25% more. It is in the resistant 25% that surgical treatment may be most justifiably considered, but even this method will not afford relief in a satisfactory number of cases at the present time (Penfield and Steelman[2]). Any new drug which can widen the scope of treatment and move some patients who have been resistant to treatment with other drugs into the favorable category merits the interest of neurosurgeons and of all other physicians who have occasion to treat chronic epilepsy.

In the course of investigation of compounds prepared in the Lederle Laboratories Division of the American Cyanamid Company, it was noted that several compounds which contained a benzylamide residue had pronounced anticonvulsant activity when tested in laboratory animals (Kushner and associates[5]). This observation led to the preparation of 40 compounds, including N-benzyl-β-chloropropionamide, or hibicon,® which proved to have a significant anticonvulsant activity, both experi-

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Read at the Fifth Congress of the Pan-Pacific Surgical Association at Honolulu, T. H., Nov. 13, 1951.

From the Department of Neurology, University of Tennessee College of Medicine. Assistant Professor of Neurology and Instructor in Neurosurgery, University of Tennessee College of Medicine.

1. Penfield, W., and Erickson, T. C.: Epilepsy and Cerebral Localization, Springfield, Ill., Charles C Thomas, Publisher, 1941.

2. Penfield, W., and Steelman, H.: Treatment of Focal Epilepsy by Cortical Excision, Ann. Surg. **126**:740, 1947.

3. Walker, A. E.: Treatment of Epilepsy by Cortical Excision, J. Pediat. **38**:285, 1951.

4. Morris, A. A.: Surgical Treatment of Psychomotor Epilepsy, M. Ann. District of Columbia **19**:121, 1950.

5. Kushner, R. I.; Casswell, J. M., II, and Williams, J. H.: Anticonvulsants: N-Benzylamides, J. Organic Chem. **16**:1283, 1951.

mentally and clinically (Gotten, Hawkes, and Tyrer,[6] Kaplan[7]). When this compound is compared with anticonvulsant drugs now in clinical use (Toman and Goodman[8]), it is readily seen to have an entirely different chemical make-up (Fig. 1). Thus, the employment of N-benzyl-β-chloropropionamide in the treatment of convulsive disorders represents a new approach to the problem.

PHARMACOLOGY

N-benzyl-β-chloropropionamide (hibicon®) is a white crystalline solid with a molecular weight of 197.6 and a melting point of 90 to 92 C. It has low solubility in water but is readily absorbed after oral administration. Experimental studies have shown that it will raise the electrical threshold in laboratory animals and will protect them against a lethal dose of pentylenetetrazole U. S. P. (metrazol®). How-

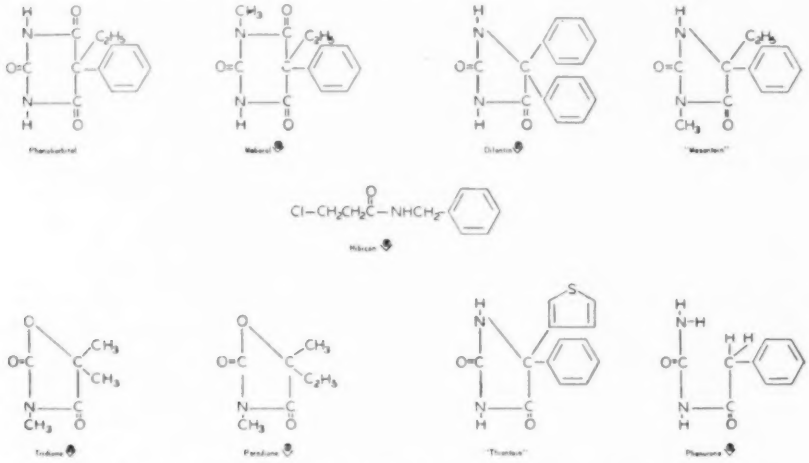

Fig. 1.—Structural formulas of hibicon® and other anticonvulsant drugs.

ever, it affords less protection against metrazol® convulsions than does trimethadione (tridione®) (Harned, Cunningham, and associates[9]).

Hibicon® has proved clinically effective in grand mal and psychomotor attacks but is ineffective in petit mal. It has a wide margin of safety, both experimentally and clinically. Side-reactions are uncommon and usually minor. The effective range of the adult dose appears to be from 1 to 2 gm. three or four times a day. In small children a dosage of 0.25 to 0.5 gm. three or four times a day has been found satisfactory.

6. Gotten, N.; Hawkes, C. D., and Tyrer, A. R., Jr.: Results in the Treatment of 54 Cases of Epilepsy with Hibicon®—a New Anticonvulsant, Tr. Am. Neurol. A. **76**:239, 1951.

7. Personal communication to the author.

8. Toman, J. E. P., and Goodman, L. S.: Anticonvulsants, Physiol. Rev. **28**:09, 1948.

9. Harned, B. K.; R. W. Cunningham, and others, to be published.

CLINICAL MATERIAL AND RESULTS

N-benzyl-β-chloropropionamide (hibicon®) has been employed by members of the department of neurology of the University of Tennessee College of Medicine in the treatment of 59 patients with chronic epilepsy during a 24-month period. This period terminated 6 months before these data were compiled, so that follow-up periods of 6 to 30 months have been provided for evaluation of the results in these patients.

In 39 of the 59 patients given clinical trial during this period, adequate control of the convulsive seizures was achieved. The drug was employed alone for all but 11 patients; for these, supplementary barbiturates were used.

In 23 of the 39 patients whose seizures were satisfactorily controlled by hibicon® the results were rated as excellent (Fig. 2). These patients were seizure-free or had

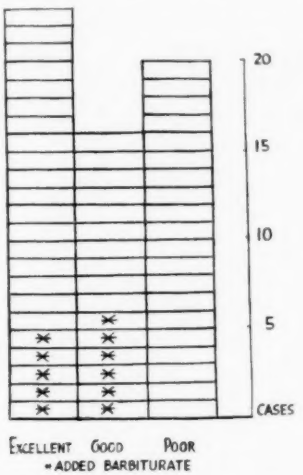

Fig. 2.—Results of clinical trial with hibicon® in 59 cases of epilepsy (6- to 30-month follow-up). The result was termed excellent when there were rare or no seizures, good when not more than one to four seizures in one month, and poor when more than one seizure in a week.

only rare attacks. In 16 patients the control was rated as good; these patients had one to four attacks a month but were nevertheless greatly benefited.

In the remaining 20 patients placed upon hibicon® therapy during the two-year trial period, the compound failed to hold the attacks below one a week, and the result was classified as poor. It is only fair to state that in eight of these patients the seizures have never been adequately controlled by any anticonvulsant drug or combination of drugs.

In the neurologic outpatient clinic of the University of Tennessee College of Medicine a comparative study was carried out (Fig. 3). Thirty-one patients were given diphenylhydantoin plus phenobarbital for trial periods, except 5 patients, for whom diphenylhydantoin was employed alone, and 7 instances in which hibicon® plus a barbiturate was given. Judged by the same standards as those cited above, 17,

or 60%, of these patients had adequate control of their seizures with the hydantoin-barbiturate combination, and 22, or 70%, had adequate control on hibicon® therapy. Excellent results were obtained with the hydantoin-barbiturate combination in 9 patients; good results, in 8 patients, and poor results in 14 patients. Excellent results were obtained on hibicon® therapy in 10 patients; good results, in 12 patients, and poor results in 9 patients. Four of the patients had adequate control of their attacks with diphenylhydantoin alone. With six patients it was necessary to supplement hibicon® with a barbiturate to achieve adequate control.

The clinicians observing the patients who had received hibicon® for its anticonvulsant activity have all been impressed by the absence of toxicity and the extremely low incidence of side-reactions which occurred with this drug. Two patients complained of gastrointestinal distress, and two, of nervousness and tremulousness. There were no serious side-reactions.

COMMENT

This clinical study demonstrates that N-benzyl-β-chloropropionamide (hibicon®) may be employed in the treatment of chronic epilepsy with an efficacy which is com-

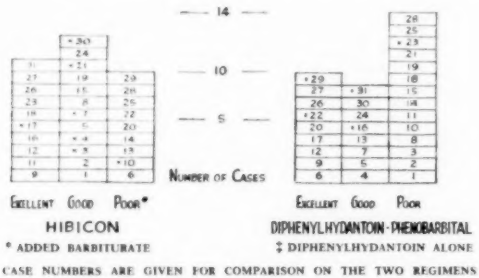

Fig. 3.—Results of a comparative study of 31 patients with chronic epilepsy from the outpatient neurological service of the University of Tennessee School of Medicine who were given clinical trials of treatment with diphenylhydantoin plus phenobarbital and with hibicon.®

The result was termed excellent when there were rare or no seizures; good when there were more than one to four seizures per month and poor when there was more than one seizure per week.

The attacks of only four patients could not be adequately controlled by either medication. Thus, hibicon® widens the scope of anticonvulsant therapy.

parable to that of other modern anticonvulsant drugs. More important, the addition of hibicon® to our present-day pharmacological armamentarium for the treatment of convulsive disorders will widen the scope of therapy. Analysis of the results in the comparative study of hibicon® and of a hydantoin-barbiturate combination shows that only 4 of the 31 patients could not be controlled by either one or the other of these modes of therapy (Fig. 3). Of the 14 patients with poor results from the hydantoin-barbiturate combination, all but 4 had adequate control of attacks with hibicon.® Of the nine patients whose attacks hibicon® failed to control, all but four had satisfactory control with diphenylhydantoin plus phenobarbital. This means that the employment of hibicon® will make possible further inroads into the resistant group of cases and give more patients adequate relief with anticonvulsant medication.

Hibicon® can be of further use in moving some patients from the group in which control is rated good into the group in which control may be rated excellent. Thus, the well-being of these patients and their economic capacity can be increased. Furthermore, the results of this study suggest that hibicon® may be the drug of choice in patients whose seizures are equally well controlled with it and with other anticonvulsant medication. The reason for this is its extremely low toxicity and the extremely small incidence of side-reactions. In the table, hibicon® is compared with other modern anticonvulsants in respect to its toxicity (Goodman and Gilman,[10] Best and Paul,[11] Lennox,[12] Little and McBryde[13]). Because of the low incidence of side-reactions, particularly the absence of sedative effect and lethargy, patients whose attacks were equally well controlled with hibicon® and with other anticonvulsant drugs frequently reported that they felt better with the new drug. Other patients, who had to take sufficient amounts of hydantoin compounds to produce

Nontoxicity of Hibicon® in Therapeutic Doses

	Drowsiness	Lethargy	Gastric Distress	Dizziness; Nervousness	Incoordination	Hyperplasia of Gums	Visual Disturbances	Agranulocytosis	Aplastic Anemia
Hibicon®.....	—	—	+	+	—	—	—	—	—
Phenobarbital.....	+++	+++	—	+	++	—	—	—	—
Mebaral® *.....	++	++	—	+	++	—	—	—	—
Dilatin® †.....	+	+	++	+	+++	+++	+	+	—
"Mesantoin" ‡.....	+	+	++	+	++	—	+	+	+
"Thiantoin" §.....	+	+	++	+	+++	+	+	+	+
Phenurone® ‖.....	+	+	+	++	+	—	+	+	++
Tridione® ¶.....	—	—	+	+++	—	—	++	++	+
Paradione® ♯.....	—	—	+	+++	—	—	++	++	+

* Mephobarbital.
† Diphenylhydantoin.
‡ Methylphenylethylhydantoin.
§ Phenthenylate.
‖ Phenacemide.
¶ Trimethadione.
♯ Paramethadione.

incoordination in order that their seizures should be controlled, had the attacks satisfactorily controlled with hibicon,® without any side-effect.

The patients in whom hibicon® has been successfully employed have been those who have had largely grand mal or psychomotor attacks, and the drug appears to be more effective in the former. Kaplan[7] and Hughes[7] have had similar experiences. In a mixed type of disorder, the addition of small amounts of barbiturate, particularly mephobarbital (mebaral®), has been found beneficial. Patients who have petit mal alone are not, in our experience, benefited by hibicon,® and this opinion is confirmed by the observations of Graf.[7] In this study hibicon® has proved equally effective in idiopathic and in symptomatic epilepsy.

10. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics, New York, The Macmillan Company, 1941.

11. Best, W. R., and Paul, J. T.: Severe Hypoplastic Anemia Following Anticonvulsant Medication, Am. J. Med. **8**:124, 1950.

12. Lennox, W. G.: Tridione® in the Treatment of Epilepsy, J. A. M. A. **134**:138, 1947.

13. Little, S. C., and McBryde, C. R.: Use of Phenylacetylurea (Phenurone®) in Convulsive Disorders, Am. J. M. Sc. **219**:494, 1950.

SUMMARY AND CONCLUSIONS

N-benzyl-β-chloropropionamide (hibicon®), a compound differing in chemical nature from other modern anticonvulsant drugs, has been found clinically and experimentally effective in the treatment of convulsive disorders.

Adequate control of convulsive seizures was achieved in 39 of 59 patients given clinical trial with hibicon,® supplemented by a barbiturate in 11. Of the 20 patients who failed to respond, 8 had never had adequate control of attacks on any anticonvulsant medication.

In a comparative study, 70% of 31 patients had adequate control with hibicon® (plus a barbiturate in 7 instances), and 60% of the same patients had control with a hydantoin-barbiturate combination.

Hibicon® has been found singularly free from toxic reactions and from untoward side-effects. Consequently, patients whose seizures are equally well controlled with this and with other anticonvulsant medication feel better on hibicon® therapy.

The employment of hibicon® appears to widen the scope of the pharmacological therapy of chronic epilepsy, since it has successfully controlled the seizures in some patients whose attacks have failed to respond to all other present-day anticonvulsant drugs.

The hibicon® for this study was supplied by Lederle Laboratories Division, the American Cyanamid Company, Pearl River, N. Y.

I wish to acknowledge the collaboration of my associates, Dr. Nicholas Gotten and Dr. Austin R. Tyrer Jr., in carrying out the clinical studies on hibicon.®

The figures illustrating this paper are taken from my scientific exhibit, "Hibicon®—A New Anticonvulsant," shown at the clinical congress of the American College of Surgeons, San Francisco, Nov.5-9, 1951.

Correspondence

NOTE ON THE HISTORY OF THE BABINSKI REFLEX

To the Editor:—In a previous note on the history of the Babinski reflex (A. M. A. Arch. Neurol. & Psychiat. **65:713**, 1951) it was pointed out that in 1893, three years before Babinski, a German neurologist, E. Remak reported the isolated dorsiflexion of the big toe on stroking the sole of the foot. The history of the Babinski reflex should contain a reference to the book of Marshall Hall (On the Diseases and Derangements of the Nervous System, London, H. Baillière, 1841). There, on page 9, Hall writes:

"I have this day (January 3, 1841) seen . . . a case of the most complete hemiplegia of sensation and volition, of the *left* side, without loss of intellect, and with the perfect continuance of all the reflex actions: respiration, deglutition, the action of the sphincter ani, are unimpaired; and the following distinct reflex actions and phenomena were observed: . . .

"On tickling the sole of the *left* foot, the extensors of the toes, and especially of the great toe, were strongly contracted; still without consciousness. The same effect was produced by applying a metallic spoon just taken out of cold, and of hot water: the extensors of the toes retained their contracted condition, whilst our visit lasted."

This was said in 1841, 55 years before Babinski.

University of California School of Medicine.

R. Wartenberg, M.D., San Francisco 22.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Hugh T. Carmichael, M.D., President, in the Chair
Regular Meeting, Feb. 12, 1952

The Tryptophan Test for Tuberculous Meningitis: A Revaluation. Dr. Joseph A. Luhan and Dr. Gilbert Lanoff.

The spinal-fluid tryptophan reaction was studied in a series of 75 patients with tuberculous meningitis. The test was performed on the spinal fluids of 275 other patients, with miscellaneous disease, for control purposes. A simple, rapid test of strong presumptive value in the diagnosis of tuberculous meningitis is highly desirable because early isolation of the organism from the fluid often fails and because other criteria, such as a low sugar content, may occasionally be lacking in this condition.

The technique of the tryptophan test of the spinal fluid was outlined, with the provision that the specimen first be centrifuged, since contaminating red blood cells might give a false-positive reaction. The occurrence of so-called false-positive reactions of xanthochromatic and grossly purulent fluids was stressed.

Positive tryptophan reactions in relatively clear, colorless fluids were obtained in 89% of cases of tuberculous meningitis by the sixth day of the disease and in over 98% of cases at some time during the course of the illness. With clinical arrest of the disease process under the influence of antibiotic therapy, the reaction became negative.

Positive reactions were obtained in dementia paralytica and in a few other conditions, unassociated with pleocytosis.

A positive tryptophan reaction with a negative Wassermann reaction and a colorless, relatively clear cerebrospinal fluid with pleocytosis is strong presumptive evidence of tuberculous meningitis.

DISCUSSION

Dr. Oscar Sugar: These results are really excellent as compared, for instance, with those of last year from one of the Swiss hospitals, in which 273 tests were run. Quantitative and qualitative studies were made, and a greater percentage of false-positive reactions was obtained. The tryptophan was not found free in the spinal fluid but came from breakdown products of the protein of the brain, which presumably had been damaged by the organism. I do not know that the criteria were the same, but certainly the results reported here are far better than those obtained elsewhere.

Dr. Gilbert Lanoff: As Dr. Sugar has stated, there has been other work on the tryptophan reaction. A recent paper by Granada and Parengan, in Italy, did not report as favorable results as ours; but, in checking their data, we found that only one test was recorded on each case, and the tests were not repeated. They also had not shown at what stage in the course of the disease the test was run. Our results have been better, also, because we eliminated false reports due to the presence of red blood cells in the spinal fluid by centrifuging the specimen and because we used the same observer for most of the tests.

A Critique of Surgery for Hydrocephalus. Dr. A. Earl Walker, Baltimore.

The techniques used in the surgical treatment of nonneoplastic hydrocephalus have been employed sufficiently long that a critical review of their effectiveness may be made. On the

basis of the medical literature and the records of the Johns Hopkins Hospital, the results at the end of two years were as shown in the tabulation.

Operation	No. of Cases	Operative Mortality	Results After Two Yr.*	
			Alive	Dead
Plexotomy	194	70	31	106
Third ventriculostomy	197	44	39	81
Arachnoidoperitoneostomy†	105	12	41	6
Arachnoidoureterostomy	86	6	17	28
Ventriculocisternostomy	25	8	9	9

* Only the patients whose states were definitely known two years after operation are coded.
† Based largely on the series of Arendt, whose results are not well documented.

While many factors enter into the analysis of these data, it would seem that for communicating hydrocephalus the peritoneal and ureteral shunts have a lower initial mortality and give a better outlook than plexotomy. For obstructive hydrocephalus, ventriculocisternostomy and ventricular shunts to the peritoneum and ureter may be more effective than third ventriculostomy.

DISCUSSION

DR. HAROLD C. VORIS: Dr. Walker has given us an interesting and informative survey of hydrocephalus and its treatment. I have always been interested in this intriguing, difficult, and often disappointing problem. I am not prepared to quote any figures, but I should like briefly to state my own experience. We have in a number of cases coagulated the choroid plexus by one of the endoscopic methods or resected it, using a modification of Dr. Dandy's technique. The results have been unsatisfactory; I have not had any cases of long-term survival in which the results were worth while. On the other hand, third ventriculostomy or a ventriculocisternal shunt has usually been successful, especially third ventriculostomy, in the group of cases of obstructive hydrocephalus associated with spina bifida and due to the Arnold-Chiari malformation. Needless to say, if either of these procedures is to be successful, there must be no obstruction of the basilar cistern, and in the case of the ventriculocisternal shunt the posterior cistern must be free of obstruction.

Ventriculoantral shunt, as suggested by Nosik, does have a distinct disadvantage of possible retrograde infection from the middle ear. However, of extracranial shunts, this is the most promising, in my opinion, at the present time. My rather sporadic attempts with ventriculoureteral and ventriculoperitoneal shunts have not met with success. As Dr. Walker has pointed out, theoretically ventriculoperitoneal shunt offers the advantage over the other extracranial shunts not only of being not subject to ascending infection but also of not producing fluid or loss of electrolytes. I do not think that Dr. Cone, of Montreal, has recorded his experience with ventriculoperitoneal shunts, but I am told that he has had considerable success. From the theoretical standpoint, ventriculoperitoneal shunt is preferable to any other peritoneal shunt. The problem, as I have already said, is an intriguing, difficult, and often disappointing one. There are sufficient successes or near-successes to constitute a challenge to us to perfect our methods to the point at which more uniform successes will be the rule. However, because of the possibility of discovering variable lesions, such as subdural hematomas, we should emphasize the importance of thoroughly studying every child with progressive enlargement of the head before cerebral atrophy produces a hopeless situation with respect to development.

DR. PERCIVAL BAILEY: My debut in neurological surgery began in the treatment of hydrocephalus when I was a student at Northwestern University. I helped Dr. Lespinasse fulgurize infants. That has been my only experience.

A New Crossed Trigeminobrachial Reflex: Anatomic and Physiologic Considerations.
DR. BENJAMIN BOSHES, and DR. DANIEL RUGE.

A patient with cervical syringomyelia exhibited a trigeminobrachial reflex. Stimulation of an area within the distribution of the left ophthalmic division produced contralateral flexion and

supination of the right forearm. The response from spastic muscles resulted from stimulation of a hypersensitive zone with either light touch or pinprick. This observation suggests that the descending tract of the trigeminal nerve reaches the fifth cervical level and contains fibers for mediation of touch, as well as pain, sensation.

DISCUSSION

DR. KRIEG: The fetal reflex referred to in the presentation is an ipsilateral reflex, but it may have a heterolateral component as well. These fibers must descend a long way before crossing, or cross at their origin, since the rachischisis was a long one. I do not know the history of the idea that only fibers for pain and temperature sense pass down the spinal portion of the trigeminal tract. Pain is relayed by the pars gelatinosa of the secondary trigeminal nucleus, which is present in both the cord and the medulla, but the pars spongiosa is a relay for touch fibers in the cord and is clearly continued into the medulla.

In the secondary trigeminal nucleus of the rat brain I have counted 11 cell groups of distinctive type. Presumably, these serve as relays for the various modalities, and the face has the best display of somesthetic modalities. This nucleus should be studied by the Marchi method.

DR. STRONG: I may be mistaken, but I think Dr. Loyal Davis had something to do with the prevalence in this region of the idea that the spinal tract of the trigeminal nerve is concerned only with pain and temperature impulses. Excellent evidence for this view, based on experimental and clinical observations, was given in an article by Margaret M. Gerard (Afferent Impulses of the Trigeminal Nerve: Intramedullary Course of Painful, Thermal and Tactile Afferent Impulses of Trigeminal Nerve, ARCH. NEUROL. & PSYCHIAT. **9**:305-338 [March] 1923).

DR. BENJAMIN BOSHES: As we were able to outline it, the zone of stimulation was exquisitely localized. We could elicit the reflex from just below the inner canthus of the left eye and the area just below the lid. This corresponds to the infratrochlear branches of the ophthalmic division of the left trigeminal nerve.

The response also was exquisite in the muscles supplied by the fibers of the fifth and sixth cervical nerves of the right side. There is no problem in understanding the crossing of the reflex, since the syringomyelia affects the corticospinal tract of the right half of the cord and there is a release on that side. But why these two segments, the fifth and sixth cervical, are picked out as levels of exquisite response is not readily answered. In accordance with previously established concepts of the distribution of the descending branch of the trigeminal nerve, it is evident that only the ophthalmic division could descend as far as the cervical area, but no studies have carried it down through the fifth and sixth cervical segments. The reflex actually appears more rapidly than was shown in the moving picture, for the latter was run slowly. There is no delay from the standpoint either of stimulation or of response. We have not attempted to measure the exact times of the stimulus and the response, but the reflex would certainly seem to be a simpler type than one mediated by intercalary neurones.

Since Dr. Walker has worked with the descending branch of the fifth nerve, I should like to hear his opinion about this.

DR. A. EARL WALKER, Baltimore: I do not have anything to contribute to this discussion. There is no question that fibers mediating touch sensation are conveyed in the descending root of the fifth cranial nerve. Sjöquist has confirmed this observation, as have a number of other investigators. I was particularly interested in seeing that the reflex described was a crossed one. I wonder whether the reflex path was via the ipsilateral descending root of the fifth cranial nerve, and then by intersegmental pathways to the opposite side, or whether the crossed secondary trigeminal pathway gave off collaterals which descended to the upper cervical segments. I would appreciate further discussion on this point.

DR. DANIEL RUGE: We wondered about the same thing. We are inclined to think that it was not the secondary pathway because of the promptness of the reflex.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY, AND NEW YORK NEUROLOGICAL SOCIETY

H. Houston Merritt, M.D., Chairman, Section of Neurology and Psychiatry, Presiding

Joint Meeting, Jan. 8, 1952

Neurological Findings in Cases of Cervical Rib. Dr. Herbert Parsons and Dr. Robert Hardy.

There is great diversity not only in the structure of cervical ribs themselves but also in associated anomalies of the subclavian artery and brachial plexus. This fact probably accounts for the multiplicity of symptoms and signs found in cases of cervical rib. In a review of 57 operations for cervical rib in 46 cases, compression of the artery was found in 23, with associated paresthesiae, color changes, aching discomfort of the extremity, and diminished amplitude of blood-vessel pulsations. There were a few instances of thrombosis and gangrene. In four cases there was an associated aneurysm of the subclavian artery. Pain and paresthesiae were the commonest symptoms, the frequency of pain in the region of the shoulder and diminishing in the more distal portions of the extremity. Paresthesiae occurred most frequently in the fingers and hand. Atrophy and weakness were found in approximately one-third of the cases, affecting chiefly the thenar eminence, and less commonly the interossei, the hypothenar eminence, and the muscles of the forearm. There were seven instances of alterations in sweating and a few of miosis and ptosis, indicating involvement of the sympathetic nervous system. In 78% of the cases cure or improvement followed scalenotomy and partial excision of the rib. Scalenotomy alone gave less satisfactory results in a small group of patients with essentially the same symptoms but without associated rib anomalies.

DISCUSSION

Dr. J. Lawrence Pool: I should like to ask Dr. Parsons in what manner he resects the cervical rib, particularly with reference to its periosteal covering, and whether in any of his cases there has been regeneration of the rib if the periosteum was left intact.

Dr. Fritz J. Cramer: Were long-tract signs associated with any of these changes?

Dr. S. Philip Goodhart: The importance of recognizing the presence of and the clinical syndrome associated with cervical rib in human beings has assumed a major place in clinical neurology in the last decade. In the majority of cases the bearer of a cervical rib is not conscious of its presence until acute symptoms appear. I believe that trauma may occasion the local changes rather acutely, but the clinical symptoms rarely manifest themselves before adulthood. Their presence is referred to as a loss of padding by reason of constitutional disease, such as anemia or apical tuberculous processes on one or both sides, so that the bony protuberance is brought into direct contact with the nerves or roots and the subclavian artery, followed by disturbances in the corresponding area of nerve supply. The authors have brought out these facts plainly.

Dr. Ira Cohen: That which is unexplained in these cases of cervical rib has always interested me more than that which is readily explained. In other words, why does the patient wait until the second or third decade of life before experiencing symptoms, and why with bilateral cervical rib does the patient have symptoms on one side only? It has been my experience that after a scalenotomy those symptoms which are directly referable to the brachial plexus clear up more readily than do those of vascular origin. I have had complete relief of the pain and of the paresthesiae, and yet there has been obvious blanching of the hand or disappearance of the pulse weeks after the operation.

Dr. Daniel Sciarra: Was headache part of the syndrome?

Dr. G. de Gutierrez-Mahoney: The authors give the impression that they advise careful exploration of the brachial plexus in all cases of cervical rib. Certainly, the problem is one of brachial neuritis, and adherents of scalenotomy alone usually do not advocate exploration of the plexus, which I think is important. May I ask Dr. Parsons whether he noted any aberrant small arteries crossing and compressing elements of the brachial plexus, such as one finds in the case of aberrant renal arteries compressing the ureter? Might it not be inferred from the figure

of 17 cases in which scalenotomy was performed in the absence of cervical rib that the authors perform scalenotomy rather infrequently as compared with the number of operations they do in cases of cervical rib?

DR. HERBERT PARSONS: In answer to Dr. Pool's question about the manner in which the rib is resected, we usually do a subperiosteal resection by the anterior approach.

As to the differential diagnosis, in a review of our cases we found two in which we were sure we made a mistake. In one of these cases a tumor subsequently developed, involving the more distal portion of the brachial plexus beneath the pectoral muscles; we did not recognize it until later. In another we did a scalenotomy because the patient had pain in the distribution of all the fingers, as well as a diminished radial pulse and decreased blood pressure, which did not improve significantly after scalenotomy. He continued to have pain, chiefly in the index and middle fingers; subsequently, we found a cervical disk between the sixth and seventh cervical vertebrae. We could never explain why the radial pulse and the blood pressure were diminished in that arm.

In no case did we find long-tract signs, although in one atrophy of one leg was associated with the anomaly; this we regarded as the residual of previous poliomyelitis. That patient may have had a lesion of the cord. Syringomyelia might be considered in this case.

We did not have many cases in which severe trauma was a precipitating factor.

With regard to atrophy of the region being responsible for pressure on a plexus, one of our patients did have tuberculosis and had had thoracoplasty, the cervical rib being left. I showed a picture of that patient.

We had several patients with pain radiating to the neck, but I do not recall any with headache.

I do not recall that we found any significant number of aberrant arteries, except as the main artery was altered.

In reply to Dr. Mahoney, we had only 17 cases of the scalenus anticus syndrome, and we did scalenotomy very much less frequently than when a definite abnormality was present. We believe that one has to be circumspect about doing a scalenotomy unless there is definite evidence of an abnormality in that region.

Group Psychotherapy with Epileptic Children and Their Mothers. DR. ZIRA DEFRIES and DR. SUE BROWDER, Stamford, Conn.

A program of group psychotherapy of epileptic children and their mothers was undertaken as part of the combined medical-psychological treatment of epilepsy. In the study reported, there were two groups of children and two groups of the mothers of these children. All the children had seizures and abnormal electroencephalograms, and all but one were receiving standard anticonvulsant therapy, which was not changed appreciably during the course of the study. In addition to the common anxieties over epilepsy, the children all had varying degrees of behavior disturbances, and the mothers presented a diversity of problems, ranging from mild anxiety over the child's seizures to deeper-seated personality problems. As a result of the group therapy, attitudes toward epilepsy were greatly improved; anxiety over the disease was notably relieved, and there was significant improvement in behavior within the group setting, as well as in school and at home. Modification of seizure frequency could not be estimated accurately because of the relatively short duration of therapy. It is believed that group psychotherapy is a useful and effective part of the treatment of the child suffering from a combination of seizures and personality disturbances and that in some instances it is more effective than individual psychotherapy.

DISCUSSION

DR. JUSTIN L. GREENE: The approach presented is one which supplies a great need in making psychotherapy available to a large number of children with epilepsy. I wonder whether in these group sessions, aside from the reactions and anxieties of the children concerned with the epilepsy, the commoner types of disturbed behavior patterns, with causes basically unrelated to epilepsy, do not tend to come out.

DR. HAROLD G. WOLFF: Will Dr. DeFries indicate how much, and in what way, she thinks the combination of therapies is better than any one alone? Is there any way of estimating the usefulness of the group-therapy procedure?

DR. ZIRA DEFRIES: In answer to Dr. Greene's question, the personality problems are quite apparent during the course of therapy, and it is our impression that some of them are altered. A significant part of the therapy was concerned with combating the more basic personality disturbances.

In answer to Dr. Wolff, the combined medical and psychiatric therapy appears to be an improvement because children whose seizures are well controlled by medication are in many instances still severely handicapped by anxieties and personality disturbances resulting from the disease. It is too early to assess fully the usefulness of group therapy. However, some of the children made much more rapid improvement with group therapy than they had made with previous individual psychotherapy.

Spinal-Cord and Local Signs Secondary to Occult Sacral Meningoceles in Adults. DR. J. LAWRENCE POOL.

Occult sacral meningoceles in adults may be associated with spinal-cord signs, which may mask the true nature of the pathologic process and thus lead to a mistaken diagnosis, such as multiple sclerosis. The progression of spinal-cord signs without remissions and their restriction to lower segments of the cord tend to rule out multiple sclerosis and other degenerative diseases.

In the two cases reported symptoms began, respectively, at 22 and 25 years of age, after trauma. Pain and sphincteric dysfunction indicated local involvement of the sacral nerve roots. Pathologic reflexes, weakness, and hyperpathia in one or both legs indicated changes in the spinal cord. In the first case the spinal cord was abnormally long, extending down through the sacral meningocele, where it was compressed by an associated lipoma. In the second case (with spastic paraparesis and a sensory level at the eighth thoracic dermatome), a double one-stage laminectomy revealed atrophy of the lower thoracic portion of the cord and a large sacral meningocele. Trauma to the latter had evidently affected the spinal cord via the filum terminale. The filum was therefore divided to release the cord from traumatic effects of traction. In the first case the lipoma could not be removed, but was well decompressed. Both patients have shown excellent and progressive improvement since operation.

DISCUSSION

DR. IRA COHEN: The second case should have been an ideal one to have produced an Arnold-Chiari syndrome, if fixation of the cord and traction are the factors in the development of that lesion.

DR. LEWIS J. DOSHAY: The pull of the filum terminale upon the spinal cord must very likely have induced some type of demyelination, rather than actual degeneration, because the process was reversible. It would seem that the recurrent impact of riding in the jeep may have damaged the myelin, rather than the nerve fibers themselves, so that after the easing of the pull there was rapid regeneration of function. This could not have occurred had there been axonal degeneration in the spinal cord.

DR. S. PHILIP GOODHART: Is there any external evidence to be found in these cases, such as a hairy nevus or changes in the skin in situ?

DR. J. LAWRENCE POOL: In reply to Dr. Goodhart's question, there were no telltale evidences of congenital anomaly of the skin, such as a hairy nevus or a pilonidal sinus, nor was there palpable or visible evidence of lipoma or spina bifida. These anomalies may be accompaniments of congenital spinal defects in that region or elsewhere in the spinal cord, but they were not present in these two cases.

In reply to Dr. Doshay, I cannot say definitely what pathologic process existed because biopsy of the spinal cord was not carried out, of course.

In reply to Dr. Cohen, there was no clinical evidence of an Arnold-Chiari syndrome in either case.

A Study of the Effects of Various Drugs Used in Treatment of Paralysis Agitans.

Dr. Harry A. Kaplan, Dr. Solomon Machover, Dr. Jefferson Browder, and Dr. Abraham M. Rabiner, Brooklyn.

This study, part of a more comprehensive investigation of the medical and surgical treatment of paralysis agitans, was undertaken to evaluate by means of objective criteria, as well as subjective measures, and by means of a carefully controlled experimental design, the relative efficacy of drugs used in the treatment of this disease. Trihexyphenidyl (artane®), caramiphen hydrochloride (panparnit®), scopolamine, and a placebo were prepared in identical capsules. Patients in four groups of 6 to 10 each were given a drug or the placebo for periods of five weeks. Each person was treated with all the drugs, and the status after treatment with each drug was compared with that following treatment with the placebo. The criteria in the evaluation of the various treatments consisted of the patients' and the relatives' reports, neurological findings, electromyographic recordings, dynamometer performance, and various measurements of psychomotor dexterity, as determined with the Purdue pegboard.

Drugs were found to be superior to a placebo in the report of an improved sense of well-being. In this respect trihexyphenidyl and caramiphen were superior to scopolamine. After a drug had been administered for the stated time, change to the placebo often resulted in complaints of discomfort. Of particular interest is the fact that patients tended to report improvement whenever a different drug was administered, regardless of sequence, although unaware of the change in medication. Even though they reported improvement in their sense of well-being, the neurological examination failed to detect any change in 57% of the patients. Despite the apparent improvement in subjective state and, to a less degree, in neurological status in response to the therapy, the electromyographic recordings indicated that no drug could reduce the amplitude of tremor as compared with the pretreatment level. On the other hand, once the patient had become adjusted to a drug, change to placebo produced, in conformity with the subjective reports of distress, a significant increase in tremor beyond the pretreatment level. Unlike the subjective reports, the neurological findings, and the myographic recordings, most of the psychomotor performances, except for the results with the dynamometer and the "shift test" on the Purdue pegboard, showed fairly constant, statistically reliable improvement after use of all drugs, including the placebo, as compared with the pretreatment level. In this respect there is a statistically unreliable suggestion of minimal superiority of drugs over placebo. The effects of the drugs are not significantly distinguishable one from the other. That psychomotor function should improve, while tremor as measured myographically does not, seems to follow from the fact that the pyschomotor test calls upon voluntary activities, which are subject to learning. Such learning, plus motivated voluntary activity, can apparently take place with about as much efficiency when patients complain of being, and actually appear to be, worse during use of the placebo, as when they are under treatment with a drug. It was noted that improvement in psychomotor function tended to be constant for each patient, and that this uniformity far overshadowed the difference between the effects of drugs and placebo.

This carefully controlled study suggests that the drugs under consideration are efficacious primarily in the subjective sphere. From the objective standpoint, no constant or reliable differential was noted between drugs and placebo.

DISCUSSION

Dr. Deso A. Weiss: My colleagues and I tried trihexyphenidyl with clinical patients, especially in view of their dysarthria. The results were often surprising. In the course of our treatment we encountered two cases that might be of interest to the neurologist. One was that of a professional boxer who had suffered such blows to his head that severe pseudobulbar dysarthria had developed. After taking the drug, he spoke rather well and fairly intelligibly. The other case was that of a woman who consulted us because of a cerebellar ataxia which affected markedly her speech as well. When she was taking trihexyphenidyl, her speech and ataxia both improved.

Dr. Lewis J. Doshay: This is an interesting attempt at measurement of changes in paralysis agitans brought on by treatment. It suggests that distinct differences are noticeable in patients during treatment with antispasmodic drugs and with placebo, at least during the short period

of the study. I am certain that if the placebo had been used consistently over a longer period the authors would invariably have discovered that the patients, instead of becoming accustomed to and happy on treatment with placebos, would all have been complaining bitterly and would have suffered a relapse in the symptoms of rigidity and tremor. Unfortunately, we have no valid tests, as yet, for the precise measurement of changes in paralysis agitans as the result of a treatment. The present authors' efforts are therefore commendable, but the validity of their tests is far from certain.

Dr. Abraham M. Rabiner, Brooklyn: Dr. Doshay seems convinced that in these cases of paralysis agitans the subjective complaints are more important than the objective evidence. Dr. Kaplan has demonstrated that by objective methods of studying motor activities it can be determined whether or not therapy has produced any improvement over the status prior to the initiation of the treatment. For many years I have maintained that the rigidity of paralysis agitans can be lessened by activity and that activity retards the tendency of rigidity to become more marked as the disease advances. All the drugs, old and new, that have been used for this disorder have some merit, and one may be a trifle more effective than others. None of them is of any value if the patient sits around and is inactive.

Dr. Solomon Machover, Brooklyn: One more point is perhaps worth emphasis. Attention was called to the fact that the minimal superiority of the drugs over placebo in producing improvement in psychomotor function is overshadowed by a trend toward individual consistency in response to treatment regardless of its nature. We measured this trend toward individual consistency by correlating the performance gains in each of the psychomotor functions under each mode of treatment with the gains in the same functions under every other mode of treatment. We found the average correlation to be in the neighborhood of 0.65. While the correlation is far from perfect, it is high enough to suggest that the patient who responds with improved psychomotor function to drug therapy is likely to respond favorably also to treatment with placebo. In the same way, patients who do not improve in psychomotor function after treatment with placebo are likely not to improve after treatment with any of the drugs.

Dr. Harry A. Kaplan, Brooklyn: There was one statement made by Dr. Doshay concerning prolonged treatment with placebo to which I should like to reply: We did carry most patients on placebo after giving them drugs. Some patients said they could not continue with the placebo capsule: However, after they were given scopolamine in small doses for a short period, they were able to continue on the placebo capsule alone.

To determine improvement of rigidity or tremor, I examined the patients personally each week. In some of them the rigidity seemed less evident on passive movement of the extremity, but they did not appear to be able to get about any better. In others there was no change in rigidity on passive movement of the extremity, but they seemed more relaxed and walked about more freely.

PHILADELPHIA NEUROLOGICAL SOCIETY

Calvin S. Drayer, M.D., *Presiding*
Regular Meeting, Feb. 1, 1952

Effect of Corticotropin (ACTH) and Cortisone on Certain Demyelinating Diseases of the Central Nervous System: Optic Neuritis. Dr. George D. Gammon, Dr. Harold G. Scheie (by invitation), Dr. Gaylord W. Ojers (by invitation), Dr. George St. G. Tyner (by invitation), and Dr. Geraldine King (by invitation).

The experiment represents an attempt to determine whether the well-known effects of corticotropin and cortisone on local inflammatory lesions in other tissues, that is, reduction of edema and of cellular exudate and scar formation, can be observed in acute lesions of the central nervous system. For the purpose, the condition of acute optic neuritis was studied because of the obvious advantage of observation of the fundus and measurements of visual fields and visual acuity. In many, if not a majority, of cases, this condition is the result of acute demyelinating diseases, in part recoverable. Evaluation therefore is difficult, and the results must be considered as a progress report rather than as fixed conclusions.

In 16 cases in which treatment was carried out, the outstanding feature was that the onset of improvement after treatment was independent of the duration of the process. Pain, visual acuity, visual fields, and edema improved, in that order, in from 1 to 10 days. This result appears to indicate a definite therapeutic effect. The final outcome, although the most important problem, cannot be compared with that of other treatments in so small a series. The actual results in this series were complete or almost complete return of vision, seven cases; partial return, four cases; no useful vision, five cases. Optic and retrobulbar lesions behaved similarly.

A response to therapy within 7 to 10 days provides a valuable yardstick for evaluation of results of treatment in lesions elsewhere, as well as in the lesions described.

DISCUSSION

DR. MICHAEL SCOTT: Was there any opportunity to use corticotropin in cases of cerebral edema not due to tumor?

DR. CALVIN DRAYER: Does this work give any lead to the possible etiologic factor in cases of less understood disturbances?

DR. GEORGE D. GAMMON: This drug has not been used in cerebral edema, that is, in the picture of pseudotumor. It does not influence and sheds no light on cause. I have three slides of the results of Landry's treatment of paralysis; I would emphasize that treatment is effective in acute lesions, and sometimes good results are obtained in chronic diseases.

Conduction of Nerve Impulses in the Regenerated Fibers of the Spinal Cord of the Cat. DR. DONALD SCOTT JR. and MR. C. CLEMENTE (by invitation).

Although Sugar and Gerard reported regeneration of spinal-cord neurons in rats (*J. Neurophysiol.* **3**:1, 1940), several authors have disputed this finding. The report by Windle and Chambers (*J. Comp. Neurol.* **93**:241, 1950) that spinal-cord regeneration could be achieved by the administration of a bacterial polysaccharide (pyromen®) was examined electrophysiologically in the present study. In one series of cats the spinal cord was sectioned, with full precautions, and the animals were allowed to recover untreated for 12 months. No sign of conduction through the site of the lesion by intrinsic spinal cord fibers was seen upon stimulation. Histological examination confirmed these results. A second series of adult cats had similar section of their spinal cords but had intravenous administration of the bacterial polysaccharide over a period of 7 to 17 months. After completion of treatment, stimulation of pyramidal tract fibers above the site of the lesion evoked impulses below the site of the lesion; these impulses could be identified by their conduction velocity as being mediated by intrinsic spinal-cord neurons. This finding was confirmed histologically. The extent to which these fibers had regenerated depended upon the administration of the polysaccharide and on the closeness of proximation of the two ends of the spinal cord at the time of the original section.

DISCUSSION

MR. C. CLEMENTE (by invitation): One of the most interesting features observed was the regeneration of facial neurons implanted in the cortex. There was glial inhibition with pyromen.® There was no glial inhibition without pyromen.® We have also found that corticotropin produces similar effects in the regeneration of spinal cord. The regenerating fibers passed through the site of the lesion when corticotropin was used, but the impulses were blocked with cortisone. We are continuing these experiments to see whether synaptic connections are made.

DR. FRANK E. NULSEN: Was any movement noted after regeneration?

DR. CHARLES RUPP: What animals were used?

DR. GEORGE D. GAMMON: We are interested in what the authors are telling us, which is, essentially, that regeneration is produced and that scar-tissue formation can be inhibited.

DR. CALVIN DRAYER: The use of pyromen® in multiple sclerosis is known.

DR. DONALD SCOTT JR.: We have thought we saw movement in the animals, but it was difficult to differentiate the spinal stepping movements from regular movements. However, if one sticks to the electrical findings, one is on solid ground. These cats were all adult.

Chronaxia as an Aid in Localization of Intraspinal Mass Lesions. Dr. Frank E. Nulsen and Dr. John Platon Kalfas (by invitation).

The strength-duration curve for a muscle, reflected in its chronaxia, will show significant alteration when lower-motor-neuron involvement is too slight to be detected on careful neurological examination. Moreover, motor weakness which is the result of pain, lack of cooperation, or disuse may be differentiated from weakness due to partial denervation.

In cases of intraspinal neoplasms, determination of segments showing denervation gives accurate knowledge as to both the superior and the inferior limit of the lesion, as demonstrated in six cases. Furthermore, in degenerative spinal-cord disease in which a mass lesion is a possibility, the presence of widespread denervation clarifies the diagnosis.

In cases of ruptured intervertebral disk with compression of a single nerve root, changes in chronaxia point to both the existence and the localization of root involvement, often not demonstrable by myelographic or clinical changes. Chronaxia changes are always of significance, and diagnosis of disk protrusion by this method has been confirmed in 17 cases. Absence of chronaxia changes is noted in rare cases in the presence of mild disk protrusion.

Comparison with the results of electromyographic studies suggests that this method is more sensitive for demonstration of slight denervation. Furthermore, many muscles can be tested in a brief examination period with minimal discomfort to the patient.

DISCUSSION

Dr. Charles Rupp: Is this a difficult or a simple technical procedure?

Dr. Joseph Yaskin: Have you retested patients after the lesion was removed?

Dr. Frank E. Nulsen: We retested only the one who had further difficulty and who was found to have rupture of a second disk.

Dr. Michael Scott: Do you feel this method will displace myelography, or must it be used in conjunction with myelography? Does it help differentiate an intraspinal lesion pressing on nerve roots and, say, a neuroma of the sciatic nerve?

Dr. Robert Groff: My only comment is that the myelogram that was reported as negative looked positive to me.

Dr. Francis Grant: I'm still not enthusiastic about flashing lights. However, when one gets an equivocal myelogram with a clinical picture and chronaxia changes indicative of an intraspinal lesion, one gets more and more confidence in Dr. Nulsen's results. So far it has betrayed us in only one case.

Dr. Abraham Ornsteen: In a case in which the herniated disk is impinging on a sensory root only, without involving a motor root, would there be a change in the chronaxia?

Dr. E. A. Denbo, Camden, N. J.: Has this method been tried in cases of obvious degenerative disease of the spinal cord?

Dr. Frank E. Nulsen: The machine is not complicated and could probably be built for $50. Anyone can be taught to run it in about a week. Another advantage in this technique is that different observers get the same figures.

Dr. Francis Grant: How long does it take to make the test?

Dr. Frank E. Nulsen: From two to three minutes for each muscle. It would take about a half-hour to check for a disk. There are not many pitfalls.

I do not think we can get along without the myelogram. The technique described amplifies the clinical and myelographic picture. So far, we have used it in cases of obvious lesions in our series. In the differential diagnosis of intraspinal and sciatic involvement, there is the matter of the changes in distribution. More muscle is involved in a peripheral lesion, with loss of sweating.

In answer to Dr. Ornsteen, it is hard to imagine a motor root not being involved to some degree in a lesion of the disk. In muscular atrophy one gets a reaction of degeneration with a very high chronaxia.

Calvin S. Drayer, M.D., *Presiding*

Regular Meeting, March 7, 1952

Observations of Effects of High Dosage Atropine Sulfate in Electroconvulsive Therapy. Dr. Rodney A. Farmer, Dr. William B. Carter (by invitation), and Dr. Lauren H. Smith (by invitation).

Fifty-nine patients were given 689 electroconvulsive treatments, and each treatment was preceded by parenteral administration of atropine sulfate in a dose ranging from 1/75 to 1/30 grain (0.86 to 2 mg.). The purpose was to modify the abruptness of onset of the convulsion, to decrease the underlying muscular tone of the convulsion, and to prevent vasovagal disturbances during the treatment. By withdrawal of atropine, at irregular intervals, the patients served as their own controls.

No instance of vagal overactivity of the heart occurred. When 1/30 grain of atropine sulfate was injected intravenously, there was a 45% chance that the onset of the convulsion would be smooth, as compared with a 10% chance without atropine. The possibility that the tone of the seizure would be low was increased from 42 to 60% with the use of atropine sulfate in a dose of 1/30 grain.

The authors present the hypothesis that convulsions occur when the cortical control is diminished and that the movements of epileptic discharges are expressions of activity or stimulation of the basal ganglia. The tonic phase of a convulsion is considered equivalent to the exaggerated rigidity of paralysis agitans, and the clonic movements are probably magnifications of the tremors of disorders of the basal ganglia. According to this hypothesis, convulsions are release phenomena rather than manifestations of cortical discharge.

DISCUSSION

Dr. Joseph Yaskin: Were there any fractures in your cases?

Dr. Rodney A. Farmer: About 5%, which is the usual rate. We were disappointed that there were any fractures.

Dr. A. Earl Walker, Baltimore: There is a great deal of discussion of the role of acetylcholine in convulsions. Atropine is antagonistic to acetylcholine. Would the authors care to discuss the possible role of atropine in the metabolism of acetylcholine?

Dr. Rodney A. Farmer: I am not prepared to discuss this problem.

Idiopathic Scoliosis in Children Due to Lesions of the Cord. Dr. Axel K. Olsen, Sayre, Pa.

Scoliosis in children is a very common and easily recognizable condition. The treatment, in the majority of instances, is carried out by the orthopedic surgeon in cooperation with the family physician. The condition is discussed as an entity in the standard texts of orthopedic surgery, and the many causes are listed. In the cases in which there is no apparent cause of the deformity, a diagnosis of idiopathic scoliosis is made and the deformity treated by manipulation, support, and, eventually, spinal fusion.

Three cases are reported, in two of which the diagnosis of idiopathic scoliosis was made and fusion carried out, when a lesion of the spinal cord was disclosed surgically; this, it is thought, was the etiologic factor in the production of the deformity. In the third case, spinal-cord disease was suspected by the orthopedist, and laminectomy was carried out before fusion was considered. The underlying disease in Case 1 was syringomyelia; in Case 2, astrocytoma of the cord, in Case 3, adhesive arachnoiditis.

It was concluded that every child with a presenting complaint of scoliosis should be studied carefully from the neurological, as well as the orthopedic, aspect. The investigation was recommended to include at least a lumbar puncture in all cases and more frequent myelograms. It was also suggested that all children with scoliosis requiring spinal fusion might well be benefited by an exploratory laminectomy at the same time.

DISCUSSION

DR. ALEXANDER SILVERSTEIN: One sees idiopathic scoliosis not infrequently. A careful neurological examination is noncontributory. I wonder what happens to the patients 3 to 10 years later. One ought to get a large series of cases from the orthopedists. I suspect that in many cases neurological signs eventually develop, as in the cases presented here.

DR. ROBERT A. GROFF: May I suggest that this paper be read before the orthopedic society?

Operation for Spasmodic Torticollis: Five Cases, Presented with Motion Pictures. DR. RUDOLPH JAEGER.

In the past 20 years operations have been performed on approximately 25 patients with more or less typical myoclonic spasms of the cervical muscles, generally known as spasmodic torticollis. Five of these patients have been followed with motion-picture records. In general, all the operations were devised with the purpose of producing paralysis or paresis of those muscles which appeared to be involved in the myoclonic activity. In some cases the muscles were removed in mass. In others the motor nerve to the muscle was sectioned. The spinal accessory nerve was cut both intrathecally and peripherally. The sensory roots were cut on occasion, but in only one of the five patients. Spinal fusion was used to stiffen the cervical portion of the spine on several occasions. Shown in the motion pictures is the technique by which the upper cervical motor roots were sectioned in one case.

All the patients treated had a prominent psychotic overlay, but whether this was the cause of their trouble it is impossible to say. It is my feeling that the torticollis is the primary disease and that the psychic abnormalities are secondary to its devastation. All the patients operated on showed definite improvement, although it cannot be said that any of them were completely cured. The contractions were decreased, the discomfort from the muscle pull was almost entirely relieved, and the mental state of the patient was greatly improved. All the five patients have returned to their original occupations and apparently are carrying on with a handicap, although at the time of the operation all of them had been absent from their usual duties for several months.

There would appear to be many reasons for attempting surgical therapy in cases of spasmodic torticollis when it is definitely resistive to psychotherapy. In those cases in which the myoclonic activity is well limited to certain muscle groups of the neck, it is believed that surgical therapy is undoubtedly superior to any other type. Not only is the intolerable myoclonia reduced, but the patient is placed in a more receptive mood for psychotherapy.

DISCUSSION

DR. JOSEPH YASKIN: It might be well to inform Dr. Jaeger that spontaneous remissions of spasmodic torticollis do occur. I am speaking of spasmodic torticollis, not the form which progresses to myotonia elsewhere. I believe that psychotherapy is as effective as surgery. The history of the treatment of spasmodic torticollis is long and colorful. Every procedure but lobotomy seems to have been tried, and sooner or later that, too, will be tried. It should not be used because most patients make a fairly good recovery with psychotherapy.

DR. ROBERT A. GROFF: I have not performed operation in many cases of torticollis myself. Dr. Jaeger is to be complimented on his good moving pictures. The operation should be bilateral. I am often unable to tell which side is involved. One should always cut the first cervical root and do a rhizotomy of the spinal accessory nerve.

DR. RUDOLPH JAEGER: I think I am entitled to disagree with Dr. Yaskin. Each of these patients came from a psychiatrist. I, too, think one should be conservative and perform this operation in stages.

The Brain Stem and Hypothalamus in Experimental Hypertension. DR. J. D. MCQUEEN, Edmonton, Alta., Canada (by invitation) and DR. A. EARL WALKER, Baltimore.

By means of a concentric electrode, placed by a stereotaxic instrument, thyratron discharges were given to the brain stem and hypothalamus of curarized dogs from which continuous blood-pressure recordings were made from the aorta by means of an electromanometer. In the diencephalon, pressor points were found in the lateral hypothalamic area, field H_2 of Forel, and

about the posterior commissure and the tract of Meynert. In the midbrain, pressor responses were obtained from the substantia grisea centralis, the lateral portion of the reticular formation, and the substantia nigra. In the superior portion of the medulla, responses were produced by stimulation of the medial and lateral reticular formations, the medial lemniscus, the dorsal and ventral nuclei of the brachium conjunctivum, and the lower portion of Deiters' nucleus. Depressor responses were of lower magnitude, less frequently found, and rarely reproducible. Anesthetic and sympathetic-blocking agents modified the response of the brain stem. The responses of the brain stem in dogs having arterial hypertension were quantitatively and qualitatively the same as those in dogs with normal pressure.

DISCUSSION

DR. ROBERT A. GROFF: How does Dr. Walker think this mechanism is initiated?

DR. MICHAEL SCOTT: Was any correlation made with attempts to increase the blood pressure by pressure on the third ventricle?

DR. A. EARL WALKER, Baltimore: Does Dr. Groff mean human responses?

DR. ROBERT A. GROFF: Yes.

DR. A. EARL WALKER, Baltimore: I do not think these changes are due to hormonal influences, for they occur in the absence of the adrenal glands. The rise in blood pressure is due to an increase in the peripheral resistance coming from a discharge from the sympathetic nervous system. This is shown by the opposite method of removing the sympathetic nervous system and getting no rise in the blood pressure. There may be a late rise, which may be due to a second renal factor. The primary factor seems to be neurogenic. We have had no experience with pressure on the third ventricle.

PHILADELPHIA PSYCHIATRIC SOCIETY

Hugo Mella, M.D., *President, in the Chair*

Regular Meeting, Feb. 8, 1952

Preverbal Aspects of Psychotherapy with Schizophrenic Patients. DR. CARL A. WHITAKER, Atlanta, Ga.

The poor integration of the schizophrenic patient results in a poor capacity to use formal symbols. This means that the verbal content of therapy with a schizophrenic patient is relatively unimportant. The problem of communication involves emotional recognition on the part of the therapist that he is being perceived (felt) and an emotional recognition on the part of the patient that he is being perceived (felt). The channels for this deeper communication include the voice characteristics which carry affect, that is, more primitive symbols, and the other methods for communicating meanings, as opposed to information. Once the communication channel is established, there will develop in both the therapist and the patient the capacity to break through these tensions and establish deeper channels, a major problem in this type of therapy. This implies a need for the deepest possible affective involvement of the therapist with the patient. Can the therapist become "crazy" about this patient? If he does, then the quality of his voice, and tensions in his facial muscles and muscles determining body posture will express this. The therapist's perception of the patient as his own child self will be communicated to the patient. The patient and his biological drive to grow is a constant. The inadequacies of the therapist as a person is the problem. These inadequacies include his inadequacy in communication. He is frequently hampered by his use of verbal substitutes for communication of affect. What does the therapist need for adequate communication with a schizophrenic patient? The capacity to use his own biological drive to grow in the framework of this relationship. This is begun by his own psychotherapy, increased by the therapy he gets from patients, and augmented by all the growthful experiences he has with colleagues. However, we must differentiate this type of personal growth from professional experience and technical growth, which is more superficial. Finally, he needs a group of colleagues under similar demands and stress, so that in this little subculture they can dare to breach the cultural barriers which so definitively disapprove of the depth of interpersonal relationship necessary for the cure of schizophrenia.

DISCUSSION

DR. MALCOLM L. HAYWARD: My thoughts are still somewhat chaotic. However, I can contribute most to the discussion by giving some more examples of situations in my experience which I have felt carried a great deal of emotional relationship between patient and therapist, and yet in which there was no actual verbal exchange. A patient was trying to describe what the therapist had done for him at the start of his illness. He said, "Assume that the patient has delusions of being Napoleon." Our analytic training tells us that the patient has killed his father and by incorporating him has become Napoleon himself. One way to deal with this is to explain to the patient, "You have killed your father, etc." When we are faced with a schizophrenic patient, the element of regression makes it difficult to do that. The patient said of his physician that simply by his presence, by his actions and personality, he conveyed to the patient the idea, "No, you are not Napoleon; I am Napoleon." The patient was freed of his guilt; the father was brought to life again, and all of this was apparently accomplished without the therapist himself being aware of it, although it was a very stormy relationship.

I also remember a woman aged 25 who for three months had been combative, cold, and distant and who suddenly, at the end of one interview, ran over to me and buried her head on my chest. The emotional impact of this was terrific. I said, "I certainly hope we can help you solve your problems." She answered angrily, "I am much too busy helping a lost lamb to find its mother to worry about that."

Another hint I got from a patient when she passed me in the hall and said out of the corner of her mouth, "Man and woman are the same: one mother," and went on her way.

In regard to the confusion made of the terms "verbal" and "extraverbal," a patient once said to me, "I never listened to your words. I watched your expression and tone of voice to see if it all added up to show love." I cannot help feeling that this is the source of a great deal of confusion in the literature. We all tend to go into considerable detail about what we said and when we said it, but what is important is how we said it and what we said besides the words.

DR. EDWARD TAYLOR: What Dr. Whitaker has said this evening is something we have all known and thought about from time to time, namely, that the treatment of a schizophrenic patient is largely a problem of the therapist. It is the therapist who always determines whether the patient is going to make progress or not. That has been my experience in my work with schizophrenic patients. When I am able to go forward myself, I find I. am usually able to bring some improvement to the patient.

Aggression is one of the major problems in the treatment of schizophrenia. I should like to mention a personal problem I had in regard to a patient with an obsessive-compulsive compulsion. I had a pretty good idea that he had a fantasy that he wanted to murder me. When I came to a point where I was not worried about what he was going to do to me, but what I was going to do to the patient, we had no problem, and we made progress.

DR. JOHN P. KOCHIS: I was getting a little panicky as the time came for me to get up and say something. Then I began to ask myself, "Why am I so confused about all this?" It goes back to something Dr. Whitaker said. I have gotten no information, but I have gotten a lot of meaning. One intense relationship with a schizophrenic patient will bring to life all that Dr. Whitaker meant. The idea that words mean nothing in such a relationship I have felt to be true many times. With the proper tone of voice and a feeling relationship with that patient, you could say, "A, B, C," and make progress with the patient. I should like to show some motion pictures we had taken of a patient at Philadelphia General Hospital. (Beginning of film shows patient getting willingly into crib type of bed.) This is a 33-year-old schizophrenic woman. The patient entered the hospital in a completely regressed state. She took intuitively to the setup we put her in. (Film continues with therapist offering a nursing bottle to the patient, now recumbent and scratching aimlessly at her head with her left thumb. She finally accepts the bottle and drinks from it while the therapist holds it.) This film was taken several months ago. By now she has somewhat lost her taste for bottle feeding. (Film continues with patient on floor displaying interest in toys shown to her by the therapist, followed by a sequence showing the patient playing with a small child and feeding him with what appears to be an apple.)

This has been an experience completely on a preverbal level. This woman had spent the previous 18 years in a psychiatric hospital. Therapy here consisted of feeling, not words. When verbal expression came into the relationship, it consisted mostly of the therapist's telling her "This is your bottle; you may have it for as long as you want, whenever you want it," and of the patient's telling what the bottle had meant to her and what the therapist had meant to her. Feeling, not words, is the most important thing we have. It is through feeling that we have given her her first 18 months out of a state hospital.

DR. KEITH FISCHER: I have had little experience with psychotic patients. Those I have seen have been in an open ward. My colleagues and I may use methods a little different, but we are interested in the same things.

I prefer the term "extraverbal" to "preverbal," which applies to something a child learns before he has learned to talk. Fromm (Forgotten Language, New York, Rinehart, Rinehart & Company, 1951) says it is a shame that there is not a language which is common to everyone; but one really does not have to look so far or create something new. There is a symbolic language. But there is one difficulty. Dr. Fromm says, "Dreams in all countries are the same." By the mere fact that we ask a person to relate a dream to us and translate it into words, we are asking him to make it verbal. This has limitations and difficulties. In our hospital we say to a patient, "If you don't want to describe the dream, go into the art room and draw about it." Very frequently, after that the patient describes it for the therapist. When, as is usual in the course of therapy, we ask the patient to tell us a dream, we are hereby asking him to take something extraverbal and translate it into something verbal.

Then, there is the acting out. The acting out that a patient does is easier to see and understand when different people are watching.

There is a language of body symptoms. It is difficult to understand and talk about. Another form of extraverbal communication comes under the heading of sublimation.

I question the idea that in therapy the patient and the therapist must have a symbiotic psychosis. We consistently refuse to have such a relationship to the patient. I should have to ask Dr. Whitaker, "Can the therapist understand the patient in his extraverbal language only by using extraverbal perceptions? And, in planning treatment, does the therapist have to have this symbiotic relationship to the patient, or is it possible that the therapist can maintain his reality functioning and, at the same time, understand the extraverbal part?"

DR. LE ROY MAEDER: It is difficult to discuss this type of presentation because some of us are working so consciously, at a conscious ego level. Psychoanalysis is an approach by analysis to psychoses from a structural and dynamic angle. We think in terms of personality, ego, superego and unconscious, but we also approach the problem from a dynamic point of view, thinking in terms of instinct, emotions, libido, fear, and the distribution of these energies in different parts of the personality and mechanisms of defense. In therapy the analyst is trying to do several things at once. He is trying to establish rapport, or empathy. He is trying to establish contact with the patient both on a feeling and on an intellectual level. He tries to find out not only the verbal, but also the affective, content. He is trying to get the patient to cooperate with him and get well.

Various people have worked on the psychoses. Freud wrote several papers on the subject. In his work with Bleuler and Jung, he was trying to adapt the libido theory to a solution of psychosis. His later work with Harry Sullivan was an effort to identify the psychotic person on all levels. I believe that the work of Freda von Reichmann is of value in an effort to understand a person on an emotional and intellectual basis. John Rosen acts things out with the patient. In this type of work, Dr. Whitaker and his associates work very much on an affective and instinctual level in the unconscious.

DR. CARL A. WHITAKER: I should like to discuss several points which have come up in the discussion. First, a word about the bottle. In the course of a year my colleagues and I have used it with 300 or 400 patients. We have used it with physicians, psychiatrists, students, and nurses. In every case we got the same type of picture.

One of the things that came out of this discussion is reflected in something that one patient told me, when I had done all I could do for her and I felt that she was getting worse and worse;

I finally said to her, "I just can't stand this any more. I have done my damnedest, had another therapist in, and we have fought it out and cried it out. To see you any more will make it worse. I shall never see you again as a patient." She came back the next week, and I told her I would not see her again. She said, "Let me tell you something. I know you have loved me, and nothing you can say or do will ever change that." That told me something: You can't fall out of love. Rejection is denial of the patient in the beginning, but one cannot reject a patient once one has accepted him.

DR. Fischer asked, "Can you learn the body language?" One of our internists always comes out of an interview with some body symptom, and it is always a symptom relating to the problem the patient has.

Someone said we act like schizophrenogenic mothers. This is perhaps true, but with one exception: We offer the patient a chance to fight free of us.

Can the therapist understand his extraverbal expression only through extraverbal means? Can one retain a sense of reality and still understand the patient? I can in those areas where I am sure and comfortable. Sometimes a patient or a medical student comes in tense and with a problem, and he can go down deep and I can retain my feeling of reality, but not with a schizophrenic patient.

We believe that when a patient regresses he is trying to satisfy a residual need at that level, and if we can free him from the need of being mothered or fathered, he will go on from there, and that his maturity is a spontaneous result of the satisfaction of the need of that moment.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Physiology and Biochemistry

BILATERAL LESIONS OF THE ANTERIOR CINGULATE GYRI. J. M. NIELSEN and L. L. JACOBS, Bull. Los Angeles Neurol. Soc. **16**:231 (June) 1951.

Within the last few years the anterior cingulate gyrus has come into prominence. Bailey and his group determined by electrical stimulation and by physiological neuronography that Area 24 is a suppressor area influencing the entire cerebral cortex. Smith's work of destroying Area 24 by undercutting or undermining produced extremely interesting results. The condition of the animals was described as "loss of social conscience." It might be described as loss of empathy.

Nielsen and Jacobs describe the sudden onset in a woman aged 46 of stupor, apathy, mutism, incontinence, inattention, and weakness of all extremities. Death occurred in one month from bronchopneumonia. Autopsy showed no material atherosclerosis in the brain but revealed embolism of the anterior cerebral arteries with secondary hemorrhage into the corpus callosum and both anterior cingulate gyri. On the right side there was a dilatation of the artery lying in the callosal sulcus, with smaller blood vessels running into it.

This patient's condition could not be called loss of social conscience; rather, it was characterized by akinesia, mutism, loss of attention, and complete apathy. It cannot be compared with any experimental cases because the monkey did not have lesions of the corpus callosum. The akinesia could be based entirely on the apathy, because complete disinterest entails complete inaction, including inaction in speech. If the apathy can then be ascribed to the lesions of Area 24, there is a fair correlation with the experimental lesions in monkeys.

ALPERS, Philadelphia.

EFFECT OF PHYSICAL ACTIVITY ON PROGNOSIS OF POLIOMYELITIS. R. M. ALBRECHT and F. B. LOCKE, J. A. M. A. **146**:769 (June 30) 1951.

This study was undertaken with the purpose of determining whether the severity of poliomyelitis could be ascribed largely to the extent of physical activity at the time of illness. It was thought that a positive relation would be of major importance for one could then prevent much of the paralysis by keeping the patient in bed during the critical period.

Two hundred patients from the 1949 poliomyelitis epidemic in Nassau County, N. Y., were studied as to their physical activity from three days before to a number of days after the onset of illness, and as to the degree of residual paralysis 5½ to 7 months after onset. It was found that death and severe paralysis in children are not related to the degree of physical activity, either shortly before the onset of illness or at any time during illness. Death and severe paralysis in adults may be attributed in part to the degree of physical activity in the meningeal stage.

Albrecht and Locke point out that age is a major factor in the prognosis of poliomyelitis in that adults, irrespective of activity, are more severely affected than children. However, factors other than age and activity are of major importance in the prognosis of poliomyelitis.

ALPERS, Philadelphia.

EFFECT OF TRANSPORTATION ON SEVERITY OF ACUTE POLIOMYELITIS. M. B. BRAHDY and S. H. KATZ, J. A. M. A. **146**:772 (June 30) 1951.

The harmful effect of physical activity on the severity of poliomyelitis was studied by Russell, who found that the amount of physical activity after onset of meningeal symptoms bore a direct relation to the degree of muscular paralysis. Hargreaves, in a similar analysis, confirmed the conclusions of Russell and showed also that the maximum exertion in one day after the onset of meningeal symptoms had a more deleterious effect on the prognosis of poliomyelitis

than the total amount of physical activity during the entire period before hospitalization. Recently, Horstmann, in an intensive study of 411 cases from three epidemic areas, confirmed the conclusions of Russell and Hargreaves.

Suspecting that long transportation might also have a deleterious effect, the authors of the present report recorded fatality rates for a group of patients with poliomyelitis who were transported to the hospital over long distances and the fatality rates for a comparable group of patients who were transported short distances. The authors' results indicate that transportation over long distances may be harmful to a patient in the acute stage of poliomyelitis. Whether the treatment of patients during the acute stage of poliomyelitis should be decentralized or whether the methods of transportation should be improved requires further study.

ALPERS, Philadelphia.

POTASSIUM DEFICIENCY IN BULBAR POLIOMYELITIS. H. S. LANS, I. F. J. STEIN, R. J. BECKER, A. L. HOYNE, and K. A. MEYER, J. A. M. A. **146**:1016 (July 14) 1951.

Paralysis of the pharyngeal musculature is frequent and troublesome in patients with bulbar or encephalobulbar poliomyelitis, resulting in difficulty or inability to swallow, associated with regurgitation. The inability to swallow necessitates intravenous and subcutaneous administration of fluids to provide nutrition and to prevent dehydration. Dehydration in patients involves not only the problem of water depletion but also that of imbalance of electrolytes. Since patients with bulbar poliomyelitis often require prolonged parenteral alimentation, the electrolyte and fluid balance of these patients becomes important.

Lans and associates studied six cases of bulbar poliomyelitis, three of which are reported here in detail. In all cases a pronounced hypopotassemia was found. Treatment of the existing deficiency with intravenously administered potassium chloride resulted in a decided improvement in the patients' condition.

Severe prolonged potassium deficiency can be fatal. This condition, when present in patients with bulbar poliomyelitis, may be a contributing factor in the high mortality rate. Proper treatment of electrolyte imbalance in these patients is essential and must include adequate potassium therapy.

ALPERS, Philadelphia.

ELECTRICAL ACTIVITY OF THE HUMAN BRAIN DURING ARTIFICIAL SLEEP. 2. REGIONAL DIFFERENTIATION OF RESPONSE TO BARBITURATE SEDATION. B. D. WYKE, J. Neurol., Neurosurg. & Psychiat. **14**:137 (May) 1951.

Wyke describes the cyclical changes induced in each of the major subdivisions of the surface of the human brain by progressive barbiturate sedation. Evidence is presented to show that during the onset of sleep the brain does not respond in over-all uniform fashion.

Wyke suggests that while in the waking state the different cortical areas of the human brain do not usually possess well-defined electrical identities, during the onset of sleep they pass through a period when they may assume more or less independent patterns of electrical behavior, the pattern of electrical activity at each stage of sleep being characteristic of the particular region.

ALPERS, Philadelphia.

LOCATION OF RECEPTORS FOR TONIC NECK REFLEXES. G. P. MCCOUCH, I. D. DEERING, and J. H. LING, J. Neurophysiol. **14**:191 (May) 1951.

McCouch and his co-workers studied the location of the receptive field for the tonic neck reflexes in a series of labyrinthectomized, decerebrate cats. Only the responses to rotation of the neck were studied in the entire series. A number of observations on the response to tilting of the head, although inadequate, suggest that their conclusions are also applicable to this reflex.

It was found that the response is retained unimpaired after section of all muscles connecting the neck or trunk with the head, after bilateral section of muscular and cutaneous branches of the first three cervical nerves, and after resection and denervation of the muscles. On the other hand, the response is abolished ipsilaterally by unilateral circumcision of the first three

cervical roots at their exit from the ligaments and is abolished in all four extremities by bilateral circumcision, although muscular and cutaneous nerves are left intact. In some animals decerebrate rigidity is lost with the neck reflexes; in others it remains unimpaired.

The authors conclude that the reflex is ipsilateral and that the receptive field lies in the region of the upper joints of the neck, especially the atlantoaxial and atlanto-occipital joints.

ALPERS, Philadelphia.

Neuropathology

NECROSIS OF THE BRAIN DUE TO RADIATION THERAPY. JOE PENNYBACKER and DOROTHY S. RUSSELL, J. Neurol., Neurosurg. & Psychiat. **11**:183 (Aug.) 1948.

Pennybacker and Russell made examination in nine cases in which necrosis of the brain followed radiation therapy. They report five of these cases, in four of which previous operation had been performed for cerebral neoplasm, while in the fifth case treatment had been given for a rodent ulcer (basal cell carcinoma) of the scalp. The diagnosis was established by biopsy in 2 cases in which recovery followed excision of the lesions, while in three cases the condition was discovered at autopsy. In the opinion of the authors, the lesions were directly attributable to effects of radiation, since no comparable findings have ever been noted to result from operation alone, nor was there any evidence of preexisting vascular disease. The pathologic changes were characterized by collagenous thickening or fibrinoid necrosis of the small blood vessels, resulting either in gradual diminution of the lumens or in thrombosis, followed by slow or rapid development of ischemia in the nerve tissue. A direct parenchymatous effect, advocated by other authors, seemed less likely. The commoner slowly progressive evolution of the vascular change corresponded to the usual long latent period (nine months to five years) noted before clinical symptoms became apparent. On the other hand, thrombosis was associated with more acute clinical manifestations. Although the incidence is unknown, such harmful effects of radiation therapy are not rare and should always be borne in mind in the case of recurrence of symptoms in cerebral neoplasms so treated.

The adjustment of the dose of radiation to prevent this complication is still to be determined. In the meantime, the authors advocate that radiation therapy be reserved for inoperable tumors.

MALAMUD, San Francisco.

HAEMANGIOMAS INVOLVING THE SPINAL EPIDURAL SPACE. A. N. GUTHKELCH, J. Neurol., Neurosurg. & Psychiat. **11**:199 (Aug.) 1948.

Guthkelch surveyed the literature relating to hemangiomas of the spinal epidural space, including those which arise from the vertebrae, and reports eight cases of his own. There are altogether 17 recorded instances of mature epidural hemangiomas and 14 cases of immature hemangioendotheliomas and hemangioblastomas. Vertebral hemangiomas, on the other hand, are usually of the mature type. They are not infrequently associated with epidural hemangiomas. Epidural tumors usually occur in the thoracic region of the spine.

Anatomically and clinically, no sharp division should be made between epidural and vertebral hemangiomas. Both are probably congenital anomalies capable of growth, the difference in type of growth depending on the activity of the surrounding tissues. Intermediate stages often occur. Both types cause compression of the spinal cord, and the clinical signs are those of progressive paraplegia with inconstant sensory changes. In a few cases, remittent attacks of paraplegia occurring during pregnancy with remission post partum were found to be caused by intraspinal hemangioma, the symptoms being precipitated by the general venous engorgement of pregnancy.

Therapeutically, however, the two types of tumor present different problems. In the case of epidural tumors, exploratory operation is necessary to establish the diagnosis; the operative results vary with the histologic structure, and the value of x-ray therapy is questionable. In the case of vertebral hemangiomas, on the other hand, the diagnosis can be made roentgenographically, and the results of x-ray therapy are generally much better than those of surgery.

MALAMUD, San Francisco.

UNILATERAL VERTEBRAL ARTERY LIGATION. L. A. FRENCH and G. L. HAINES, J. Neurosurg. **2**:156 (March) 1950.

French and Haines report a case of ligation of the left vertebral artery which terminated fatally with thrombosis of the basilar artery.

A white man, aged 48, had an 11-year history of progressively developing dizzy spells, occipital headaches, visual hallucinations, and disturbances in gait. At the time of his admission all the deep reflexes were hyperactive, and he presented a Hoffmann sign on the left, decreased right abdominal reflexes, and slurred speech. A ventriculogram showed a defect in the left occipital horn. An extremely vascular meningioma, arising from the tentorium, was encountered at operation. Total extirpation was impossible, owing to the vascularity of the tumor. Because of the progression of symptoms, the tumor was again surgically attacked two months later. Seven months after this operation a vertebral angiogram was obtained on the left side; this procedure was followed by a rapid downhill course, terminating in death three days after ligation.

Autopsy showed thrombosis of the entire left vertebral artery and of the basilar artery up to 2.5 cm. above the origin of the superior cerebellar artery. There was softening of the pons on each side of the basilar artery, being more pronounced on the left side. The meningioma had involved the greater part of the left cerebellar hemisphere and had extended supratentorially into the parieto-occipital area. The cause of death was considered to be the infarction of the pons.

TOZER, Topeka, Kan.

SUBACUTE NECROTIZING ENCEPHALOMYELOPATHY IN AN INFANT. D. LEIGH, J. Neurol., Neurosurg. & Psychiat. **14**:216 (Aug.) 1951.

Leigh reports the case of a boy aged 7 months who died within six weeks of a disorder of the central nervous system characterized by somnolence, blindness, deafness, and spasticity of the limbs.

The neuropathologic picture consisted in focal, bilaterally symmetrical, subacute necrotic lesions or softenings extending from the thalamus to the pons, the inferior olivary nuclei, and the posterior columns of the spinal cord. Histologically, the affected areas showed an intense vascular, microglial, and histiocytic proliferation and a less pronounced astrocytic increase. There was severe neuronal damage in these areas.

In discussing the etiology and pathogenesis, Leigh suggests that an unknown toxin or virus was responsible for these lesions or, more probably, that these lesions represent the reaction of an immature brain to the same type of nutritional deficiency which produces the Wernicke syndrome in the adult.

ALPERS, Philadelphia.

Psychiatry and Psychopathology

BEHAVIOR DISTURBANCES IN EPILEPTIC CHILDREN. C. BRADLEY, J. A. M. A. **146**:436 (June 2) 1951.

Bradley reports his observations on behavior difficulties in intelligent and physically intact children subject to convulsive seizures. These behavior disorders are of two general sorts: There are often behavior symptoms which are the primary, direct result of the neurophysiologic disturbances that may be recorded electroencephalographically, and that are also presumably directly related to the seizures themselves. These behavior symptoms consist of five component features: (1) erratic variability in mood or behavior, (2) hypermotility, (3) irritability, (4) short and vacillating attention span, and (5) a rather selective difficulty in mathematics as a school subject. Bradley states that such behavior symptoms are best treated medically, the most effectual preparations at present available for this purpose being the amphetamines.

Other behavior disturbances, representing the child's reaction to being ill and to the way he is handled by those about him, being subject to more variables than the primary symptoms, do not fall into any such characteristic pattern; these are less specific in nature and must be treated at the psychologic level. Thus, adequate medical management of the epileptic child involves consideration of his behavior and emotional reactions, in addition to therapy designed to control his seizures.

ALPERS, Philadelphia.

PSYCHOSIS FOLLOWING "AMATEUR HYPNOSIS": CASE REPORT. I. S. DRIBBEN, Mil. Surgeon **104**:136 (Feb.) 1949.

Dribben presents the case of a youth aged 17 who had been recently inducted and who was brought to the hospital in a state of acute excitement, with complete detachment, incoherence, confusion, and disorientation. A group of enlisted men had been attempting mesmeric hypnosis for amusement, and one of them had succeeded in hypnotizing the patient but had been unable to "bring him back." After a short period in the receiving office, the patient became less excited, and contact was restored, but he remained amnesic in respect to his own identity and to the incidents immediately prior to his hypnosis. The following morning there was a recurrence of the amnesia and confusion. He was then taken by his "buddies" to another hypnotist, who attempted mesmerism with constructive suggestions. This produced temporary improvement. The same night, when he was brought back to the hospital, he was lethargic, detached, disoriented, confused, and amnesic, particularly in regard to the hypnosis episode. Auditory hallucinations were noted. The lethargy and mild detachment persisted. He acquired an insight into the psychotic episode and believed that it was caused by the hypnosis. Later he was separated from military service. This youth, who was constitutionally handicapped and had a schizoid diasthesis, reacted with psychosis to hypnosis induced by unqualified "amateur" mesmerists.

J. A. M. A.

EFFECTS OF STRESS AND THE RESULTS OF MEDICATION IN DIFFERENT PERSONALITIES WITH PARKINSON'S DISEASE. JOHN S. PRITCHARD, ROBERT S. SCHWAB and WILLIAM A. TILLMANN, Psychosom. Med. **13**:106 (March-April) 1951.

Pritchard, Schwab, and Tillmann, who are supervising the treatment and general management of patients with Parkinson's disease (paralysis agitans), noted that favorable responses to treatment and unfavorable responses to stress of various kinds differ greatly from patient to patient. The purpose of the present investigation was to analyze these differences and to attempt to relate them to the personality of the patient concerned.

One hundred patients with Parkinson's disease were studied as to their personality type. Pritchard, Schwab, and Tillmann divided these patients into the following groups: (A) normal, stable, easy-going personalities, 48 patients; (B) suggestible, dependent personalities, 33 patients; (C) driving, restless, assertive personalities, 19 patients.

It was found that Group A had a low incidence of exacerbation of disease due to stress (12%), Group B had an incidence of 21%, and Group C, the highest incidence (58%).

Both Groups A and B responded favorably to medical therapy (79 and 77%), whereas only 37% of Group C responded favorably. Observations of this kind enable the physician to make a better prognosis in the treatment of a patient with Parkinson's disease by understanding the personality type.

GUTTMAN, Wilkes-Barre, Pa.

PHYSIOLOGIC AND PSYCHODYNAMIC MECHANISMS IN CONSTIPATION AND DIARRHEA. T. S. SZASZ, Psychosom. Med. **13**:112 (March-April) 1951.

Szasz, in examining this problem, attempts an elucidation of some basic methodologic principles in the psychosomatic approach to lower gastrointestinal dysfunctions.

The author gives the following summary: Constipation and diarrhea are symptoms related to disorders of the lower portion of the gastrointestinal tract. In the past, little consideration has been given to the precise portions of the tract involved in these symptoms.

The lower portion of the alimentary tract is divisible into two distinct functional units: that of the colon and rectum, which are regulated by the autonomic nervous system, and that of the external anal sphincter, which is under voluntary control in consequence of previous toilet training. Disorders of the colon and rectum express themselves either in increased secretion and motility, resulting in diarrhea, or in a decreased activity, resulting in constipation. Disorders of the anal sphincter are best analyzed in terms that are applicable also to the vesical sphincter, viz., retention, frequency, and incontinence. Thus, constipation may be the result of an increased retention, and diarrhea, the result of an increased frequency. Whereas the disorders of colonic function are related to the dynamics of the vegetative neuroses, the dysfunctions of the anal sphincter are related to the dynamics of hysterical conversion symptoms.

The usual psychoanalytic formulations concerning constipation and diarrhea are inadequate because they can account only for dysfunctions of the anal sphincter. A new hypothesis of the physiologic basis and of the psychodynamics of colonic dysfunction is presented. It is suggested that colonic activation and inhibition, leading to diarrhea and constipation, represent the physiologic sequelae of a certain alteration in the upper gastrointestinal tract. Constipation is regarded as the remote physiologic consequence of a state of increased vagal excitation, and diarrhea is interpreted as the result of a sudden decrease in vagal tonus. Psychologic factors of etiologic significance in colonic dysfunctions are related to mobilization and inhibition of oral-incorporative tendencies. The symptoms themselves which result from colonic dysfunctions have no primary psychologic meaning.

GUTTMAN, Wilkes-Barre, Pa.

Meninges and Blood Vessels

DISSECTING ANEURYSM OF THE AORTA: TWO CASES WITH UNUSUAL ONSETS. C. FELDMAN, Ann. Int. Med. **35**:728 (Sept.) 1951.

Feldman describes the clinical picture and course of dissecting aneurysm of the aorta and discusses the various concepts of the pathogenesis, the pathologic changes, and the relation of the intimal tear to the dissection. He reports two cases in which the onset was unusual.

The onset in the first case was atypical, since pain did not play a major role. Blurring of vision and headache were the presenting complaints. Signs and symptoms of pericarditis were the outstanding features from the second day to the seventh, the day of sudden death, from cardiac tamponade. The clinical diagnosis was pericarditis. There was a history of hypertension. Autopsy did not reveal any intimal tear, and the dissection involved only the portion of the aorta within the pericardial sac.

The second case began with pain, situated primarily in the episternal notch, where severe tenderness and a systolic thrill were elicited. A diastolic murmur was present from the start. A correct diagnosis was made ante mortem. Death from massive cardiac tamponade was sudden. The blood pressure was somewhat elevated in this case. An intimal rent was present, and the dissection involved the entire aorta and the common iliac arteries, where the aneurysm emptied itself into the arterial lumen, thus forming a "double-barreled aorta."

ALPERS, Philadelphia.

TUBERCULOUS MENINGITIS CURED WITH STREPTOMYCIN: REPORT OF A CASE COMPLICATED BY RENAL AND URETERAL CALCULUS. J. T. AQUILINA, J. A. M. A. **147**:560 (Oct. 6) 1951.

Aquilina reports a case of bacteriologically proved tuberculous meningitis in an American Indian who recovered after a long course of streptomycin therapy. The diagnosis of tuberculous meningitis was established by culture and guinea-pig inoculation. The patient was alive and well three years after the onset of his illness.

The treatment consisted of 350.8 gm. of streptomycin administered intramuscularly over a period of 168 days and of 6.9 gm. administered intrathecally over a period of 85 days. Calculus formation was observed as a complication, necessitating pyelolithotomy and nephrostomy on the right side and, later, nephrectomy on the left. The only residuals of tuberculous meningitis were blindness in the left eye and slight weakness of the left leg.

ALPERS, Philadelphia.

SPINAL MENINGITIS DUE TO ACTINOMYCES BOVIS TREATED WITH PENICILLIN AND STREPTOMYCIN. C. EDWARDS, W. A. ELLIOTT, and K. J. RANDALL, J. Neurol., Neurosurg. & Psychiat. **14**:134 (May) 1951.

The involvement of the central nervous system by actinomycosis is rare. It may arise in one of three ways: (1) by primary infection, (2) by direct extension, and (3) by metastasis. The advent of antibiotics has greatly changed the prognosis of all varieties of the disease.

The authors review the literature on actinomycotic meningitis and describe a case of meningitis, limited to the spinal region. It was probably of the primary type, no other focus having been discovered, despite careful search. The disease was treated and cured by penicillin and streptomycin.

ALPERS, Philadelphia.

NEUROLOGIC AND METABOLIC EFFECTS OF BILATERAL LIGATION OF THE ANTERIOR CEREBRAL ARTERIES IN MAN. C. S. MACCARTY and I. S. COOPER, Proc. Staff Meet., Mayo Clin. **26**:185 (May 23) 1951.

The effects of bilateral ligation of the anterior cerebral artery have been disputed in medical literature for several years. Dandy stated that ligation of both anterior cerebral arteries in man, close to the site of their origin, is incompatible with life. Poppen, however, said that either one or both of the anterior cerebral arteries can be ligated without causing loss of consciousness if the blood pressure is kept within normal limits while the ligation is being performed. Experimental studies by Watts support Poppen's conclusions, and recently Thompson and Rhode reported that pathologic unconsciousness does not occur in monkeys surviving the immediate postoperative period after ligation of the anterior cerebral arteries at their point of origin. They stated that neurologic sequelae roughly parallel the degree of peripheral hypotension when the anterior cerebral artery is occluded.

This report is based on a case in which both anterior cerebral arteries of a human being were ligated. The observations made in this case substantiate, to a large measure, the results of the experimental studies of Watts, and of Thompson and Rhodes. During the postoperative period, examination discloses no pronounced focal neurologic abnormalities, and the patient was lucid and mentally clear on several occasions. Moreover, during these lucid intervals, the patient was relatively free of gross neurologic abnormalities. It is important to note that the systemic blood pressure was 120 mm. systolic and 80 mm. diastolic at the time the anterior cerebral arteries were ligated.

There also were periods in which hypotension, stupor, and muscular flaccidity were present in the postoperative period. Marked electrolyte abnormalities persisted from the fifth postoperative day until the patient died, on the 40th day after ligation of the anterior cerebral arteries. These consisted of hypernatremia, hyperchloremia, and hypochloruria.

These electrolyte abnormalities appear to have contributed to the death of the patient. The relation of this unusual electrolyte picture to the cerebral lesions in this case is not clear, although it is possible that the metabolic abnormalities were sequelae of the cerebral lesions.

Necropsy revealed remnants of the adenoma of the pituitary body, for which the patient was operated on, and evidence of subtotal resection of the right frontal lobe of the brain and bilateral ligation of the anterior cerebral artery at the site of the anterior communicating artery. Massive infarction was present in both frontal lobes and in both corpora striata. The significant microscopic changes in the brain were massive infarcts of both frontal lobes and both corpora striata and smaller infarcts situated throughout the brain, but most numerous in the region of the left hippocampus.

ALPERS, Philadelphia.

PERSONALITY PATTERNS IN PATIENTS WITH MALIGNANT TUMORS OF THE BREAST AND CERVIX: AN EXPLORATORY STUDY. M. TARLAU and I. SMALHEISER, Psychosom. Med. **13**:117 (March-April) 1951.

Tarlau and Smalheiser report on a pilot study which was undertaken to determine whether there are similar personality patterns in two groups of cancer patients: women with cancer of the breast and women with cancer of the cervix uteri. The sample consisted of 22 married women, 11 with cancer of the breast and 11 with cancer of the cervix. The age range was from 27 to 58. The mean age of the group with breast cancer was 50, and they had been ill an average of 32.6 months, while the mean age of the group with cervical cancer was 48, and the average length of illness was 31.2 months. The general educational level of the group was that of finishing grade school. Since they were patients in a city hospital, the socioeconomic backgrounds of the two groups can be considered similar.

Three techniques of investigation were used: a personal interview, lasting from one to two hours: the Rorschach method of personality diagnosis, and drawings of the human figure. The interview was directed toward those factors which influence the psychosexual development of the patients. It included the early family life, the age of the patient at the parents' death, sex education and reaction to menstruation, and the area of marital adjustment. The authors state that the Rorschach data were the most valuable, for while the patients may distort interview

material, their functioning is revealed clearly in their reaction to the unstructured ink blots. The drawings of the human figure were used to supplement and corroborate the diagnosis arrived at by the Rorschach test.

The authors state that the interview material and the Rorschach data gave consistent pictures for the two groups. The differences in the life histories of the patients with breast and the patients with cervical cancer, although the two groups came from a similar socioeconomic background, were indicative of definite differences in the behavioral patterns of the two groups. Both groups had similar family patterns of mother dominance, which resulted in a rejection of the feminine role. Both groups had similarly negative attitudes toward sexuality, which contributed to sexual maladjustment. The group with breast cancer was able to adjust superficially to these conditions. Sexual conflict was not overt in this group. These women had no premarital experience; they married at a late age, and their marriages were stable. The stability of their marriages, however, is not to be interpreted as meaning a happy marriage. Their marriages were overtly stable because of the lack of demands by their partners.

In the patients with cervical cancer, on the other hand, the sexual disturbance was much more overt. There were a higher incidence of premarital relations, a lower age at marriage, and a very high incidence of overt marital discord. The signs of disturbance were also evident in the test results. The authors state: "From our data we conclude that the breast cancers appeared in women who were functioning psychologically at a primitive oral level, while cervix cancers appeared in patients who had a genital fixation. These personality patterns revealed by the Rorschach data are probably not the result of the cancer process itself. The existence of these problems previous to the illness is revealed by the interview material. We feel that there is some evidence here which suggests that the personality structure may play a role in the pathogenesis of cancer of primary or secondary sex organs in predisposed individuals.

"This study is only a preliminary one, being limited to two small groups of cases of similar social background; the findings must face the test of further study on larger groups of patients with cancers of other body organs and in patients with non-malignant diseases of the same organs."

GUTTMAN, Wilkes-Barre, Pa.

Diseases of the Brain

PARKINSONISM AND RHEUMATOID ARTHRITIS. R. O. WALLERSTEIN, Ann. Int. Med. **34**:899 (April) 1951.

In an effort to evaluate critically the relation between the two diseases, 19 patients with frank clinical paralysis agitans and 28 patients with far-advanced, chronic atrophic arthritis were examined.

Certain superficial similarities of the hands were noted. Absence of true joint disease by clinical or roentgenographic evidence was demonstrated in patients with Parkinsonism. Only one patient (in this group of 500 patients) appeared to have both diseases. The author concludes that there appears to be no increasing incidence of rheumatoid arthritis among patients suffering from Parkinsonism.

ALPERS, Philadelphia.

DIENCEPHALIC EPILEPSY AND THE DIENCEPHALIC SYNDROME. H. MANDELBAUM, S. D. SPATT, and L. E. FIERER, Ann. Int. Med. **34**:911 (April) 1951.

The authors present a case of diencephalic epilepsy due to mumps encephalitis. The patient exhibited a wide range of emotional symptoms, including rage and libido. Vasomotor instability and autonomic functional disturbances were manifold. These included skin changes, perspiration, wide fluctuations in blood pressure, irregularities of heart rhythm due to premature contractions, changes from hypothermia to low-grade fever, diabetes insipidus, occasional glycosuria, colonic irritability, and dysmenorrhea and menorrhagia. Somatic tremors recurred frequently. Sedation proved of no value. A large dose of estrogen resulted in establishing a remission.

Mandelbaum and his colleagues point out that the diencephalic syndrome is fairly common. It embodies the characteristic emotional, vasomotor, and autonomic disorders described above. Being dissatisfied with the results in such cases of restriction of sodium, sedation, and psycho-

therapy, adequate doses of estrogen were employed, and the symptoms were controlled. Moreover, the treatment program with estrogen was effective in delaying the appearance of sustained hypertension in many of these patients.

In 52 cases demonstrating marked lability of blood pressure, the piperoxan (benzodioxan) hydrochloride test was used. It proved useful in differentiating cases of diencephalic syndrome from those of pheochromocytoma.

ALPERS, Philadelphia.

HEREDITY OF EPILEPSY AS TOLD BY RELATIVES AND TWINS. W. G. LENNOX, J. A. M. A. **146**:528 (June 9) 1951.

In a study of the 20,000 near relatives of 4,231 epileptic patients, a history of seizure was obtained in 3.2%. The incidence was 3.6% if evidence of brain damage prior to the patient's first seizure was lacking, and 1.8% if such evidence was present. These percentages are, respectively, 7 and 3.5 times the incidence of epilepsy among draftees of the United States Army. As additional evidence of a genetic factor, in the group with undamaged brain (essential epilepsy) the incidence of epilepsy among relatives decreased progressively with a later onset of seizures (7.6% if onset was in infancy; 1.5% if it was after the age of 30). Corresponding incidences for the group with brain damage (symptomatic epilepsy) were 2.9 and 1.3%.

The relative influences of heredity, brain damage, and chronicity of seizures were studied in a group of 122 twin pairs affected with seizures. In twin pairs without prior brain damage, both co-twins were epileptic in 84% of the one-egg twins and in 10% of the two-egg twins. In pairs with brain damage, the corresponding incidences were 17 and 8%. In one-egg twins, concordance was usual not only with respect to seizures but also with respect to the type of seizures and to the electroencephalographic pattern. Intelligence quotients were determined for 87 twin pairs. The person's mental endowment was of most importance in the maintenance of a good intelligence. Acquired brain damage was a severe depressant; the reported bad effect of repeated seizures was not in evidence.

The genetic factor in epilepsy is probably no greater than it is in many other common diseases. The epileptic is unfairly pillorized in public opinion and in law. Assets that are transmissible, such as sound vital organs, good intelligence, personality, and social responsibility, may outweigh the liability of a tendency to seizures. Hence Lennox feels that advice regarding marriage and children must be individualized.

ALPERS, Philadelphia.

SUBACUTE CORTICAL CEREBELLAR DEGENERATION AND ITS RELATION TO CARCINOMA. W. R. BRAIN, P. M. DANIEL, and J. G. GREENFIELD, J. Neurol., Neurosurg. & Psychiat. **14**:59 (May) 1951.

A review of the literature shows a high correlation of carcinoma with subacute degeneration of the cerebellar cortex. Of cases which have run their course in two years, carcinoma has been present in more than two-thirds, although this has not always been clinically evident. The more chronic forms of cerebellar degeneration show no such correlation.

In this report, Brain and his colleagues describe four cases of subacute degeneration of the cerebellar cortex, in three of which there was also degeneration of the long tracts of the spinal cord (direct spinocerebellar tract in all, the pyramidal tract in one, and the dorsal columns in the other two). Of the three cases reported, there was also carcinoma of the bronchi in two and of the ovary in one.

Progressive dementia appeared soon after the onset of cerebellar symptoms in three of the four cases. Diplopia of the cerebellar type was present in all cases and was an early symptom in three. Cramp-like pains in the legs heralded the onset of cerebellar symptoms in two cases.

The authors believe it is clear that this is not a sharply defined pathologic syndrome or disease entity but in rare cases in which degeneration of Purkinje cells come on rapidly, sometimes associated with degeneration of related systems and tracts, especially the spinocerebellar tract and the superior cerebellar peduncle. In the majority of these subacute cases carcinoma appears to play an etiologic part, either by special alteration of the body metabolism or in combination with some factor at present unknown.

ALPERS, Philadelphia.

ALTERATIONS OF CEREBRAL FUNCTIONS IN PHEOCHROMOCYTOMA. N. S. APTER; W. C. HALSTEAD; A. S. ALVING; P. J. TALSO, and T. J. CASE, Neurology **1**:283 (July-Aug.) 1951.

Reports of tumors arising from chromaffin tissue of the adrenal gland (pheochromocytoma) and from ganglia of the sympathetic nervous system (paraganglioma) are being reported with increasing frequency. The pressor substances from these structures discharged into the general circulation produce effects that are of special interest to the neurologist and the psychiatrist. The clinical picture resembles that of anxiety states, vasovagal attacks, and hypothalamic disorders. The syndrome consists of paroxysmal or sustained hypertension, palpitation, sweating, pallor, tremulousness, headache, dyspnea, nausea and vomiting, pain in the precordium and epigastrium, and muscular weakness. Also encountered are vasomotor phenomena, such as discoloration of the hands and legs or blanching of digits, as well as hypermetabolism, glycosuria, amenorrhea, changes in urinary frequency, loss of libido, and hyperthermia.

Apter and his colleagues describe combined medical, neurologic, experimental psychologic, electroencephalographic, and psychiatric studies done before and after operation on a patient aged 14 with pheochromocytoma. These studies revealed that temporary impairment of functions of the cerebral cortex takes place. The generalized systemic vascular changes are accompanied by similar alterations in the cerebral blood vessels and are reversible. These investigators suggest that the somatic experience of anxiety is dissociated from consciousness by virtue of temporary dysfunction of the cerebral cortex.

Preoperative and postoperative electroencephalographic readings showing the effects of piperoxan hydrochloride and epinephrine on the tracings are also recorded.

ALPERS, Philadelphia.

ELECTROENCEPHALOGRAPHIC FINDINGS IN POST-TRAUMATIC ENCEPHALOPATHY. A. A. WEIL, Neurology **1**:293 (July-Aug.) 1951.

Electroencephalographic tracings of 50 patients with post-traumatic encephalopathy were correlated with the clinical findings. It was noticed that the abnormality of the electroencephalogram increased with the severity of trauma; there were a definite increase of abnormal electroencephalograms (83%) and a significant increase of focal electroencephalographic abnormalities (83%) in patients who retained neurologic abnormalities; 41% of patients without neurologic abnormalities showed irregularities of the electroencephalogram indicative of an organic lesion.

ALPERS, Philadelphia.

ELECTROENCEPHALOGRAPHIC FOCI IN TEMPORAL LOBES WITH CONSIDERATION OF SEIZURE MANIFESTATIONS. C. W. IOBST and F. M. FORSTER, Neurology **1**:309 (July-Aug.) 1951.

This report embodies the observations on a series of 50 consecutive patients presenting focal dysrhythmias from the temporal lobe in electroencephalograms. The dysrhythmias appeared spontaneously in 52% of the group, a higher incidence than might have been expected.

Iobst and Forster point out that when the dysrhythmia in the routine tracing is not sufficiently clear-cut or when, from the clinical viewpoint, a temporal-lobe origin is suspected, a record must be obtained during sleep. The clinical factors suggesting a temporal-lobe origin are (*a*) the presence of psychomotor seizures and (*b*) the presence of atypical attacks or of major convulsions preceded by factors suggesting, on the basis of cortical localization, a temporal-lobe origin. According to these criteria, records obtained during sleep established the presence of the temporal-lobe focus in 61% of this group.

When the clinical situation suggests a temporal-lobe origin of the patient's seizures and when both spontaneous records and sleep records are normal, pentylenetetrazole (methazole®) activation should be employed. By resorting to this, the authors found an additional three cases in their series.

Etiologic factors which produced discharging lesions from the temporal lobe were head injury, birth injury, brain tumor, electric shock, spontaneous subarachnoid hemorrhage, and dementia paralytica.

It has been emphasized in recent years that psychomotor seizures are the epileptic manifestations of lesions of the temporal lobe. In the present series only 48% of patients with temporal-lobe foci presented psychomotor seizures.

The nature of the electroencephalographic focus suggests the possibilities of medical therapy. If a disease process for which surgical therapy is mandatory can be excluded, the patient with a temporal-lobe focus has available both medical therapy and elective surgical intervention. Patients in this series whose temporal-lobe dysrhythmias appeared spontaneously were more amenable to medical therapy than were patients requiring activation.

ALPERS, Philadelphia.

PHENOMENA AND CORRELATES OF THE PSYCHOMOTOR TRIAD. W. G. LENNOX, Neurology **1**:357 (Sept.-Oct.) 1951.

On the basis of clinical characteristics, Lennox divides the psychomotor (temporal-lobe) epilepsies into three groups and eight subgroups. In a study of 414 patients, seizure types were distributed among the three groups as follows: psychomotor, 43%; automatic, 32%, and subjective (psychic), 25%. The relative sizes of the automatic and subjective groups increased progressively with increasing age of the patients.

Of 1,900 office patients, 20% gave a history of psychomotor (temporal-lobe) seizures. With increasing age there was an increase both in the incidence of psychomotor seizures (relative to other types) and in the number of patients having both psychomotor and grand mal seizures. Sixty-three per cent of all psychomotor patients had a history of grand mal also, but only 3% had a history of petit mal. In the grand mal-psychomotor group, grand mal attacks began a year or more before psychomotor seizures in 64% and a year or more after psychomotor seizures in 36%. Acquired cerebral lesions antedated the onset of epilepsy in 33% of patients having both convulsions and psychomotor seizures and in only 15% of patients having only one of the petit mal triad. The incidence of epilepsy among near relatives was 2.7% for patients having psychomotor seizures (alone or associated with grand mal), but it was 5.4% for those with a history of petit mal and grand mal.

These data suggest that psychomotor seizures may arise as a consequence of damage to a temporal lobe, possibly as a result of earlier convulsions, anoxia, trauma, or infection.

ALPERS, Philadelphia.

INTRACRANIAL NEOPLASM ASSOCIATED WITH FOURTEEN AND SIX PER SECOND POSITIVE SPIKES. W. A. STEPHENSON, Neurology **1**:372 (Sept.-Oct.) 1951.

The clinical correlates of 14- and 6-per-second positive spikes have been reported by Gibbs and Gibbs, who concluded that such spikes are of thalamic or hypothalamic origin. Stephenson encountered 32 such patterns in 2,500 adult patients. Relatively few of these patients showed evidence of organic disease of the nervous system. In the two patients who are the subject of this paper, neoplasms were verified at operation, and the anatomic location was such as to afford support to the thesis that the source of the positive spikes was in the diencephalon.

In the first patient, electroencephalographic and isotope studies at first failed to demonstrate the tumor, which proved to be subcortical. Incomplete removal was carried out as deep as the ependyma, and after eight months there was some evidence of progression. At that time sleep recordings taken soon after a grand mal seizure showed characteristic 14-per-second positive spike discharges appearing on the right side from temporal, parietal, and occipital derivations. It is believed that the positive spikes were an irritative response to the extension of the tumor medially to the central gray masses.

In the second case, the only abnormal feature of the electroencephalogram was the presence of 6-per-second positive spike discharges during sleep. It was noted at operation that there was invasion of the third ventricle. Such invasion would impinge on the diencephalic areas which are thought to be the origin of 14- and 6-per-second positive spikes.

From the records on these two patients with proved intracranial neoplasms involving the deep nuclear structures, Stephenson concludes that these structures are the source of the 14- and 6-per-second positive spikes.

ALPERS, Philadelphia.

ATROPHY OF THE OPTIC NERVE DUE TO SCLEROSIS OF THE INTERNAL CAROTID ARTERY. P. PARIN, Schweiz. Arch. Neurol. u. Psychiat. **67**:139, 1951.

On the basis of experience with three cases and a review of the literature, Parin discusses the symptoms, pathogenesis, differential diagnosis, and treatment of atrophy of the optic nerve due to compression of the nerve by a sclerotic internal carotid artery. The relation of the two structures is such that when the artery is either enlarged or abnormally tortuous it is capable of pressing the nerve against the fold of dura which forms a backward prolongation of the roof of the optic canal.

The first patient, a woman aged 48, suddenly lost the vision in her left eye 13 days after beginning to experience nausea and a feeling of pressure behind both eyes. Examination revealed bilateral papilledema, graded 2 D. on the left and somewhat less on the right; vision of 1/50 in the left eye and 1.0 in the right, and inferior altitudinal hemianopsia on the left side. Central vision and the defect in the visual field both quickly improved, along with subsidence of the papilledema. General examination disclosed evidence of cardiovascular disease and beginning cerebral arteriosclerosis. In the course of the next 18 months, vision in the left eye became temporarily worse on six occasions. Despite the transient periods of improved vision, optic nerve atrophy developed on the left, and the inferior altitudinal defect in the left visual field gradually became more extensive. Although x-ray examination of the skull revealed no abnormalities when the patient was first seen, stereoscopic lateral projections taken 13 months later showed calcification of the left internal carotid artery.

The other two patients were men in their late 70's who obviously had general and cerebral arteriosclerosis. In each case roentgenologic study of the skull revealed calcification of the internal carotid artery on the side of visual failure. In the first of these two patients the left eye became blind in the course of a few hours, during which period the patient observed a gradual constriction of his left visual field both from above and from below. An ophthalmologist who examined the patient at this time found nothing remarkable in the affected eye. Examination by the author five weeks later revealed optic nerve atrophy with reduction of vision to light perception on the left side. The third patient suddenly discovered he was blind in the right eye. Examination 20 days later revealed slight edema of the optic disk of the right eye with reduction of vision to light perception in this eye. Optic nerve atrophy soon developed in the affected eye.

DANIELS, Denver.

Diseases of the Spinal Cord

HYPOVENTILATION SYNDROME IN BULBAR POLIOMYELITIS. S. J. SARNOFF; J. L. WHITTENBERGER, and J. E. AFFELDT, J. A. M. A. **147**:30 (Sept. 1) 1951.

The gross irregularity of breathing and the ventilatory inadequacy which may be associated are part of the classic picture of bulbar poliomyelitis. When a clear airway has been insured and the respiratory defect persists, the latter is then attributable to involvement of the respiratory center. The nature of the defect may be apparent clinically in two ways: (1) gross irregularity of the rate and rhythm of respiration and (2) incoordination of the various muscles of respiration. In addition, a third, and perhaps more important, type of respiratory defect occurs.

This third type of respiratory defect in bulbar poliomyelitis, which the authors term the hypoventilation syndrome, is the subject of this paper. It consists of a decreased sensitivity of the respiratory center to the partial pressure of carbon dioxide in arterial blood. This results in lowered pulmonary ventilation, which is attributable to a diminished respiratory drive and in some ways is analogous to the situation in which the respiratory center has been depressed by an overdose of barbiturate.

Four patients with bulbar poliomyelitis were seen whose respiration was such as to include them in the category of the hypoventilation syndrome. It was found that verbal artificial respiration can be effectively administered to the patient if he is responsive and is observed continuously by competent personnel members, and if it is not required for too prolonged a period.

The administration of oxygen may possibly be hazardous under these conditions. The potential hazards, however, may be readily overcome if any significant diminution of spontaneous ventilation is reversed by some means of artificial respiration.

Electrophrenic respiration may be of help under circumstances in which spontaneous respiration is inadequate or becomes so after the administration of oxygen. At least one phrenic nerve must be uninvolved by the disease if electrophrenic respiration is to be used.

ALPERS, Philadelphia.

Peripheral and Cranial Nerves

SHOULDER-HAND SYNDROME FOLLOWING MYOCARDIAL INFARCTION: TREATMENT BY PROCAINE BLOCK OF THE STELLATE GANGLION. D. M. SWAN and J. M. MCGOWAN, J. A. M. A. **146**:774 (June 30) 1951.

Reflex dystrophy of the upper extremity, often producing serious disability, has received increased attention in recent years. Although the syndrome has many causes, the underlying neurovascular mechanism is probably the same. This paper is concerned with a description of a technique for procaine block of the stellate ganglion by the anterior approach and the results obtained in treatment of the shoulder-hand syndrome following myocardial infarction.

The shoulder-hand syndrome occurs in approximately 10 to 20% of cases of myocardial infarction. The degree of disability reported has been variable; in some cases it has been described as minor; in others it has been extremely severe, leading to serious incapacity and inability of the patient to perform useful work. Formerly, treatment recommended for this condition included physical therapy, x-ray treatments, ganglionectomy, and periarterial sympathectomy. A sufficient number of cases of procaine block of the stellate ganglion have been reported in the literature to indicate the beneficial results of this treatment.

Swan and McGowan here report the results of treatment of a series of patients with the shoulder-hand syndrome by stellate block with procaine hydrochloride by the anterior approach. Within 10 minutes after injection there was complete relief of pain in the shoulder and arm, and in one case there was also relief of a pain in the precordial region which the patient had complained of constantly. More striking, even, than the relief of pain was the remarkable return of function in the muscles of the extremity. This method is considered by the authors to be fairly simple and is consistently successful when employed with a reasonable amount of skill. Their results were excellent in every case.

ALPERS, Philadelphia.

A CASE OF GUSTATORY SWEATING. H. I. TANKEL, J. Neurol., Neurosurg. & Psychiat. **14**:129 (May) 1951.

Profuse facial sweating on eating is a rare phenomenon, of interest not only as a clinical problem but also as a means of elucidating the complex mechanism of the neural and hormonal control of perspiration. Of the cases described hitherto, the sweating in all but one occurred as a result of sympathetic paraylsis, particularly in the distribution of the auriculotemporal nerve. The present case is unusual in there being no such predisposing cause and in its presenting unusual features on investigation.

The patient, a man aged 44, was admitted for gastrectomy after a history of duodenal ulcer for 27 years. Incidental to his history of ulcer was the complaint of sweating on the left side of his face on eating for the past three years. The sweating was gradually increasing in severity. There was no history of injury or disease either local or referable to the central nervous system, but 10 or 12 years before the onset of the sweating the patient had suffered from sensitivity to sunlight, which produced vesiculation of the forehead. This reaction to sunlight had not occurred since the onset of gustatory sweating.

There was no Horner syndrome; the pupils were equal, and no abnormality of the central nervous system was detected. The skin temperature was the same on the two sides. A roentgenogram of the skull and cervical vertebrae showed calcification of the pineal gland, choroid plexus, and posterior clinopetrous ligaments. The Wassermann reaction of the blood was negative.

In an investigation of the afferent pathways, it was confirmed that sweating occurred in response to the stimulation of taste fibers by food and that the response was proportional to the intensity of the stimulus. The pathway is therefore considered to be by way of the taste fibers of the glossopharyngeal nerve and the chorda tympani component of the facial nerve.

On investigation of the effector mechanism, it was found that gustatory sweating depends on the integrity of fibers in the supraorbital nerve and the cervical sympathetic chain and that it is inhibited by atropine. There was no reason to suggest that these fibers are other than the normal sweat fibers.

In an effort to demonstrate any other abnormality of sweating in the gustatory sweating area, it was found that the left side of the face was more sensitive to the action of heat, acetylcholine, and pilocarpine than the right side. Whether the glands themselves or another part of the gustatory reflex arc is sensitive remains to be decided.

Since a sensitivity of some sort appeared to be present, it was decided on empirical grounds to try the effect of an injection of histamine. In a normal subject histamine does not produce sweating except as a side-effect. Sweating cannot be produced in a normal or a sympathectomized area of skin by local injection, nor does a normal or a sympathectomized limb sweat when histamine is injected intra-arterially.

The notable features in this case are that histamine produced sweating and that this sweating was produced only in the gustatory sweating area. The accurate localization of the sweating suggests that histamine or a similar substance plays a part in the gustatory sweating reflex arc. A further feature in support of this argument was the finding that various antihistamine drugs used by Tankel inhibited the gustatory sweating in proportion to their antihistamine activity.

The pathology of this abnormality is unknown.

ALPERS, Philadelphia.

Treatment, Neurosurgery

EXPERIMENTAL AND CLINICAL INVESTIGATIONS OF PERIPHERAL NERVE INJURIES OF THE UPPER EXTREMITIES. G. PERRET, J. A. M. A. **146**:556 (June 9) 1951.

Early recognition of a peripheral nerve injury is the greatest single prophylactic measure that can be taken to insure a good result after surgical repair of an interruption in the continuity of a nerve trunk. In the presence of a peripheral nerve injury, the sooner the ends of the divided nerve can be united in accurate apposition with the finest suture material and the most meticulous technique possible, the more successful will be the surgical end-result.

If immediate end-to-end suture is not possible because of loss of nerve substance, it is advisable to identify the proximal and distal nerve ends and anchor them as closely together as possible to surrounding muscle, fascia, or tendons with wire sutures. Thus, they will be more easily found when definite repair of the nerve gap is undertaken.

To effect any result in a peripheral nerve injury in which there is loss of substance such that an end-to-end apposition cannot be obtained, the following four possibilities may be considered: (*a*) use of an autogenous graft; (*b*) use of homogenous graft; (*c*) shortening of the limb, and (*d*) fixation of joints and transposition of tendons of uninjured muscles.

Traumatic lesions that involve parts of the brachial plexus are not uncommon in civilian or military life. The results of the surgical treatment of brachial-plexus lesions are, on the whole, not as good as those of peripheral nerves. The distance between the site of the lesion and the muscles and area of skin involved make nerve regeneration a slow and prolonged process.

Finally, Perret states that the most important principle affecting the results of peripheral nerve surgery, and one that is not sufficiently emphasized, is that principle that stresses every effort to keep the effector mechanism in a state in which it may function. Consequently, the intensive use of physical therapy and all its adjuncts—electrical stimulation, massage, active and passive movements, and use of proper splinting of the paralyzed extremity into a position of relaxation of the paralyzed muscle and of function of the limb—is of great importance and cannot be overemphasized.

ALPERS, Philadelphia.

CLINICAL EVALUATION OF ACTH AND CORTISONE IN MYASTHENIA GRAVIS. C. H. MILLIKAN and L. M. EATON, Neurology **1**:145 (March-April) 1951.

Five patients with myasthenia gravis consented to receive one or more courses of injections of cortisone or corticotropin (ACTH). In every patient increased muscular weakness was observed toward the end of each course of cortisone or corticotropin. Two patients had no improvement; one patient had slight transitory improvement; one patient had moderate transitory improvement, and one patient had decided transitory improvement after completion of the courses of cortisone or corticotropin.

Because of the increase in symptoms during the last part of a course of corticotropin or cortisone, caution must be used when administering these substances to myasthenic patients. Patients with great weakness may become worse to the point of decompensation of respiratory function and to the point at which neostigmine will not provide an adequate margin of safety. One of the patients in this series died while undergoing a course of cortisone therapy, and a second died suddenly three months after the completion of a course of cortisone therapy.

Until time permits further study of the three surviving patients, the evidence is that cortisone and corticotropin are of limited value in the treatment of myasthenia gravis.

ALPERS, Philadelphia.

News and Comment

THIRD INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY

The Third International Congress of Electroencephalography and Clinical Neurophysiology will meet in Boston, Aug. 18 to 21, 1953.

Since Berger's classic description of the human electroencephalogram, in 1929, and Adrian's confirmation of this work, published in England in 1933, the clinical interest in electroencephalography has grown tremendously throughout the world. Enough clinical units existed in England in 1945 that a national society of electroencephalography was founded in that country. In the United States the first society was founded in 1946—the Eastern Association of Electroencephalographers and, later the same year, the national society, the American Society of Electroencephalography, was founded. At the present time there are three additional regional societies in the United States. There are national societies in France, Norway, Netherlands, Switzerland, Spain, and an international South American Society.

In 1947, the first international congress in this field met in London, and the International Federation of EEG Societies was formed. This organization is incorporated in Paris and is affiliated with UNESCO. The international journal of *Electroencephalography and Clinical Neurophysiology* stems from this meeting and is now in its third year. The second international congress was held in Paris in 1949.

Related fields of clinical neurophysiology, such as electromyography and various experimental techniques involving animals, have been closely associated with clinical electroencephalography.

The third international congress will continue for approximately a week, and we hope to have 500 delegates attending this meeting. There will be general symposia on such subjects as epilepsy and other nervous and mental diseases. In addition to the symposia, there will be a large number of individual presentations, the usual panels and exhibits, and opportunities for commercial exhibits of both technical apparatus and pharmaceutical products which would be of interest to this group. The American Branch of the League Against Epilepsy, Inc., will meet at the same time in Boston, and other specialized groups may also collaborate.

The great difficulty, of course, is to make possible a large attendance from outside the United States. The international committee expects the secretary-general to raise a fund of between $20,000 and $30,000, which will be used to partially aid the foreign delegates to attend the conference. Our committee is working in liaison with a similar one in supporting the International Physiological Congress, which meets the first week of September, 1953, in Montreal.

The American Society of Electroencephalography, as a start, has donated the sum of $500 toward this fund. We hope to set up four types of financial support for foreign members attending this Congress.

1. Unrestricted gifts of money to the general travel fund of the Congress, which would be used by the officers of the Congress to help the travel expenses of foreign members requesting aid. For example, the officers might provide a scientist from Sweden with half his return steamship passage if he requests aid. Another scientist from a different country might require a larger or smaller grant to enable him to come. We would try to arrange it so that the actual decisions as to those helped would stem from individual committees in the countries concerned, subject, of course, to the approval of the Congress officers.

2. Specific travel grants to attend the Congress limited to scientists in particular countries, or in particular fields of endeavor, that the contributing pharmaceutical company chooses to set up. For example, a Swiss drug company might care to arrange for a travel grant to be used by a Swiss scientist either known to them or applying through one of their representatives. They might also request that such a grantee of their financial support would include a separate trip to the American branch of the company during his visit to the United States.

3. Specific travel grants of different amounts turned over to the committee and identified by name with the drug concern to be awarded to scientists chosen by the Congress officers.

4. United States Government and Rockefeller Foundation travel grants for travel in this country, which would be awarded at a time that would enable the recipient to attend this Congress, and probably the one in Montreal.

At present we are not certain as to how the various pharmaceutical houses would like to have travel grants identified, but this could be worked out in more detail later. We realize that this is somewhat early to negotiate funds, but preliminary approaches in a study of the funds available for support in relation to actual numbers of foreign members who need it is important.

In the August 1951 issue of *Electroencephalography and Clinical Neurophysiology* there was a notice of the Congress printed with a form to be filled out by all delegates who plan to attend. In the form there is a statement as to whether attendance is possible without financial help from the Congress. We expect to know exactly within a few months in this regard, but at present we have estimated that we shall need between $20,000 and $30,000 to take care of the various requests that are coming in and will be expected in the subsequent year.

RESIDENCY TRAINING IN PSYCHIATRY, VETERANS ADMINISTRATION

An approved three-year residency training program in psychiatry is available at the new Veterans Administration Hospital in Seattle. This training program is under the direct supervision of the department of psychiatry at the University of Washington School of Medicine, of which Dr. Herbert S. Ripley is professor and chairman of the department.

In the absence of a university hospital, this particular Veterans Administration Hospital is active in fulfilling the needs of the medical school in that regard. Affiliated with the training program at the Veterans Administration Hospital is an arrangement by which the resident can obtain experience in the outpatient department at the King County Hospital, the Child Guidance Clinic of the University of Washington School of Medicine, the Veterans Administration Hospital at American Lake, and the Mental Hygiene Clinic.

This hospital has 100 beds allotted to neurology and psychiatry and provides an adequate number of patients for an excellent resident training program. The city of Seattle also offers opportunity for analytic training to qualified persons.

The salary range is that of the Veterans Administration residency training program, from $2,400, for the first year of residency, to $3,000, for the third year.

CONGRESS OF NEUROLOGICAL SURGEONS

The second annual meeting of the Congress of Neurological Surgeons will be held at the Palmer House, Chicago, on Nov. 6, 7, and 8, 1952. Among the speakers will be Prof. Herbert Olivecrona, of Stockholm, Sweden; Dr. Albert Kuntz, professor of anatomy and director of the Department of Anatomy, St. Louis University School of Medicine; Dr. E. A. Hines, Mayo Clinic, Rochester, Minn.; Dr. Francis L. McNaughton, assistant professor of neurology and neurosurgery in the McGill University Faculty of Medicine, and Dr. Harold Wolff, Cornell University Medical College, New York.

The Congress of Neurological Surgeons was organized a year ago with the express objective of attracting to membership those neurosurgeons less than 45 years of age, although older neurosurgeons are welcomed to the group. There is no numerical limitation on membership.

Requirements for membership provide only that the applicant shall have either (1) completed the formal training prescribed for entrance to examination by the American Board of Neurological Surgery, or (2) practiced neurosurgery for a sufficient period of time to have gained such professional esteem as to distinguish him as a competent practitioner who consistently does creditable work.

In neither instance is certification by the American Board of Neurological Surgery necessary for membership in the Congress of Neurological Suregons.

The organization is designed to present the postgraduate type of educational program to the membership. Recognized leaders in the field will be asked to participate in the programs of the group.

Neurosurgeons interested in membership may communicate with Dr. Bland W. Cannon, secretary, Congress of Neurological Surgeons, 1092 Madison Avenue, Memphis, Tenn.

ISAAC RAY MEDICAL LIBRARY

On March 5, 1952, the Isaac Ray Medical Library was dedicated and opened at Butler Hospital in Providence, R. I. Around a valuable nucleus of books on medical psychology from the libraries of Isaac Ray, M.D., G. Alder Blumer, M.D., and other former superintendents, it is hoped that an unusually good working collection will eventually be assembled, totaling up to 10,000 volumes. So that the library will be equipped to meet more than local needs, it is planned to compile a union catalog of holdings of other psychiatric libraries in this country, making the library a valuable research instrument for psychiatrists elsewhere.

The project was conceived by Dr. Gregory Zilboorg, consultant in research and psychotherapy at Butler Hospital, and received generous support from the Aquinas Fund. Its new and attractive quarters were designed and built entirely by hospital personnel.

At the dedication, Dr. Leo Bartemeir and Dr. Gregory Zilboorg were the principal speakers.

POSTGRADUATE COURSE IN PSYCHIATRY AND NEUROLOGY, UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE

Ten Weeks, August 25th through October 31st, 1952

The postgraduate in psychiatry and neurology will be given from Aug. 25 to Oct. 31, 1952, at the Langley Porter Clinic. This course is given by the Division of Psychiatry, University of California School of Medicine, in cooperation with University Extension (Medical Extension), University of California, and is a repetition of the course given previously. This course is open only to qualified physicians.

Instruction will be given under the direction of Dr. Karl M. Bowman, professor of psychiatry, University of California School of Medicine, with the assistance of staff members from the various divisions of the medical school.

Hours will be Monday through Friday, 9 a. m. to 5 p. m., with some special work on Saturdays, 8 a. m. to 10 a. m., for part of the time.

Subjects to be covered will include general psychiatry; child psychiatry; psychobiology; psychoanalysis; psychology and psychopathology; functional and organic psychoses; psychoneuroses; therapy; psychosomatic problems; neuroanatomy; clinical neurology; neuropathology; neurophysiology; electroencephalography; x-ray diagnosis; cultural anthropology, and related topics.

The course is particularly designed to prepare psychiatrists and neurologists for taking the examinations of the American Board of Psychiatry and Neurology. It is, therefore, designed for the advanced student in psychiatry and neurology, rather than the beginner. A special endeavor is made to present the latest knowledge and advances, so as to make the student familiar with the most recent developments in psychiatry and neurology.

The fee for this course is $200, payable in advance by check or money order made to The Regents of the University of California. Fee should be included with application and biographical data, as follows: (1) place of legal residence; (2) medical school attended and year of graduation; (3) training, and experience in psychiatry. *Application, fee and biographical data* are to be sent to STACY R. METTIER, M.D., Professor of Medicine, Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22. Further details may be obtained from the office above or from The Langley Porter Clinic (address above).

University Extension reserves the right to cancel this course, in which case all fees will be refunded. This course will not be given for less than 25 registrants.

AMERICAN PSYCHOSOMATIC SOCIETY

At the annual business meeting of the American Psychosomatic Society, held on March 29, 1952, the following persons took office: Sydney G. Margolin, M.D., president; George L. Engel, M.D., president-elect; Fredrick C. Redlich, M.D., secretary-treasurer.

Elected to council positions were David T. Graham, M.D.; George C. Ham, M.D.; Erich Lindemann, M.D., and Milton Rosenbaum, M.D.

SOCIEDAD DE NEUROLOGIA Y NEUROCIRUGIA

The Sociedad de neurología y neurocirugía of Montevideo, Uruguay, has elected the governing board for 1952 and 1953; Dr. Bernardino Rodríguez, president; Prof. Agdo. Román Arana Iñiguez, vice president; Dr. Kempis Vidal Beretervide, secretary; Dr. Lorenz Perez Achard, prosecretary, and Dr. Elio García Austt Jr., treasurer.

GERMAN SOCIETY FOR NEUROLOGY

The German Society for Neurology, jointly with the German Society for Physiological Chemistry and the German Society for Neurosurgery, will hold its annual meeting in Hamburg, Sept. 25 to 27, 1952. The main subjects for discussion are metabolism permeability of the nervous system; edema, and aneurysms, including angiomata of the brain. Communications may be addressed to the secretary, Prof. G. Döring, Krankenhaus Heidberg, Hamburg-La 2.

Books

Psychosomatic Gynecology, Including Problems of Obstetrical Care. By William S. Kroger, M.D., Assistant Clinical Professor of Obstetrics and Gynecology, Chicago Medical School; Attending Obstetrician and Gynecologist, Edgewater Hospital, Chicago, and S. Charles Freed, M.D., Adjunct in Medicine, Mount Zion Hospital, San Francisco. Pp. 503. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1951.

This comprehensive and encyclopedic volume also includes an extensive section by Dr. Grantly Dick Read on the psychosomatic aspects of pregnancy.

Dr. Kroger is a gynecologist-obstetrician psychoanalytically oriented. Dr. Freed is an endocrinologist. The authors state that "our interest in psychiatry arose from personal experiences and the conviction of a great need for an understanding of psychodynamics so as to give proper service to our patients. Perhaps, because of lack of orthodox training, this volume may actually prove of greater aid than otherwise to the average physician and indirectly, to the psychiatrist."

This pioneering work should be of inestimable value to all gynecologists and obstetricians whether they agree with the conclusions of the authors or not. Each section contains an extensive bibliography, and each chapter is followed by many references. A valuable section is the glossary, chiefly of psychiatric terms.

Many gynecologic disorders are influenced by the psyche, frequently through the endocrine secretions, and the authors have closely followed the investigations of Benedek, Rubenstein, and Wolfe and the monumental work of Selye. The psychiatric references are complete and are not restricted to one group. The sources cited include Freud, English, Dunbar, Mandy, Deutsch, Fenichel, Alexander, Menninger, and many others.

It is patently impossible in one volume to discuss fully all of the psychodynamic factors associated with gynecologic complaints, but every phase is covered and the text is replete with case histories, which are extremely helpful.

Unfortunately, at the present time, there is a dichotomy between psychiatry and gynecology. Many gynecologists are unaware of the emotional origin of symptoms and by accepting them at their face value, instead of helping, perpetuate neurotic conflicts.

Although every gynecologist cannot be psychiatrically trained, this volume should give him insight into the psychodynamics of his specialty.

The authors have made a very real contribution, and, in this reviewer's opinion, their book marks an epoch in the correlation of psychiatry and gynecology.

Précis de psychiatrie. By Henri Baruk. Price, 1,600 fr. Pp. 614. Masson & Cie, 120 Boulevard Saint-Germain, Paris 6e, 1950.

In this textbook of psychiatry, Dr. Baruk, who is best known here for his monograph on mental symptoms associated with brain tumors and his investigation of bulbocapnine experimental catatonia in animals, presents a summary of 25 years' clinical experience. The point of view is that of "synthetic psychiatry," with discussions of all that is known about the human personality from the biologic, clinical, psychologic, social, and moral aspects. Mental ailments are described not as clinical entities but as psychologic and biologic reactions to varied physical and "moral" stresses. It is a complete work except for the omission of psychiatric problems of childhood. This book would have been impossible without the cooperation of many of the author's students, who contributed much while working on their dissertations.

The author considers psychosomatic medicine, as the term is used in this country, a misnomer and believes it should be called psychogenic or psychoanalytic psychiatry, for it is really a search for psychogenic causes of all sorts of ailments. The term "psychosomatic" is used by Baruk in a different sense, to describe in great detail the efforts to study the physiologic alterations accompanying mood changes and mental illness with utilization of all available techniques. In no other summary of psychiatric knowledge is there such a detailed description of somatic

and physiologic concomitants of mental changes. Vestibular and electromyographic changes in catalepsy, capillaroscopic alterations in depression, toxicity of bile in mental affections, oscillometric records in hysteria, and hormone assays of body fluids in psychoses are discussed in a lively and interesting text. The approach is stimulating and provocative, and the text is frequently spiced by allusions to personal clinical experience. The reviewer approves strongly of the repeated references to the author's own clinical experience. These enrich the text and fortify the writer's point of view. A few of the more unusual case reports are worth mentioning —bradycardia and fatal gastric bleeding after forced feeding, hallucinations referable to the chest associated with malignant neoplasm of the mediastinum, disappearance of writer's cramp after removal of a toxic goiter, Lilliputian hallucinations accompanying a tumor of the hypophysis, and perineal neuralgia as a depressive equivalent.

As a result of physiologic and biologic study of the psychoses and neuroses, the author points out some interesting correlations which merit the attention of other investigators. Ideas of jealousy were noted as related to hepatic function, and paranoid ideas, to vascular congestion of the brain. Tuberculous lesions were observed to be affected by mental changes. The role of colon bacillus infections in depressions is also emphasized.

The significance of psychogenic factors is not overlooked. The point of view of the Freudian school is criticized for its interest in the happiness and freedom from anxiety of the individual and its neglect of social and "moral" factors. Baruk emphasizes the psychogenic role of guilt. He accounts for paranoid tendencies, hate, prejudice, and neurotic tendencies by what he calls the Tsedek complex, describing a test for this complex which measures the "moral sense." In this theory of the role of the guilt complex in the psychogenesis of mental ailments, the author was apparently influenced by Hebraic thought, and especially by the teachings of the prophets.

The chapters on practical and administrative psychiatry are very valuable. Nowhere has the reviewer encountered such clarity in outlining the problem of the relation between psychiatrist and patient. This should be read not only by every young psychiatrist but also by the more experienced for a salutary point of view.

The statement about the probable effect of alcohol on the germ plasm is surprising and is not supported by any evidence. In spite of the author's emphasis on organic factors in mental disease, there is no mention in the text of the Korsakoff syndrome with subarachnoid hemorrhage, paranoid reactions with pernicious anemia, or schizophrenic-like syndromes in bromide intoxication. Nothing is mentioned about the relation of postpartum psychosis to manic-depressive psychosis. The discussion of paranoia as a character psychosis is unclear because no attempt is made to define the term "character."

The use of biblical quotations and Hebrew idioms to justify the author's theory about psychosomatic relationships hardly appears justified in a work of this type. The danger of such a philologic argument is evident to any student of the history of ideas. The coinage of these words was not based upon any presaging of modern theories, and certainly not upon scientific reasoning. It is also difficult to understand Baruk's opinion that the great religious teachers were not psychotic even if they did hallucinate. The theory that hallucinatory experiences can be physiologic and that intense exaltation and faith can produce experiences smacks of apologetics. The attitude that the contributions of the religious leaders are less significant because they were psychotic was attacked pithily by William James when he reiterated the well-known "Judge them not by their roots but by their fruits."

Baruk makes no mention of amphetamine (benzedrine *) for the treatment of depression and minimizes the value of penicillin in dementia paralytica. His attitude toward all forms of shock therapy is hostile. He emphasizes the significance of isolated severe complications of shock therapy without objective evaluation. He mentions dementia following shock therapy, definitely unknown in our experience. His condemnation of psychosurgery is also unjustifiable. He recommends psychotherapy and autoserotherapy for hypomania and transcerebral calcium dielectrolysis for depression. It is difficult to understand why bromides with low-salt intake should be prescribed for manic patients.

In spite of minor faults, this provocative book, which is replete with fruitful concepts, is a valuable addition to psychiatric literature.

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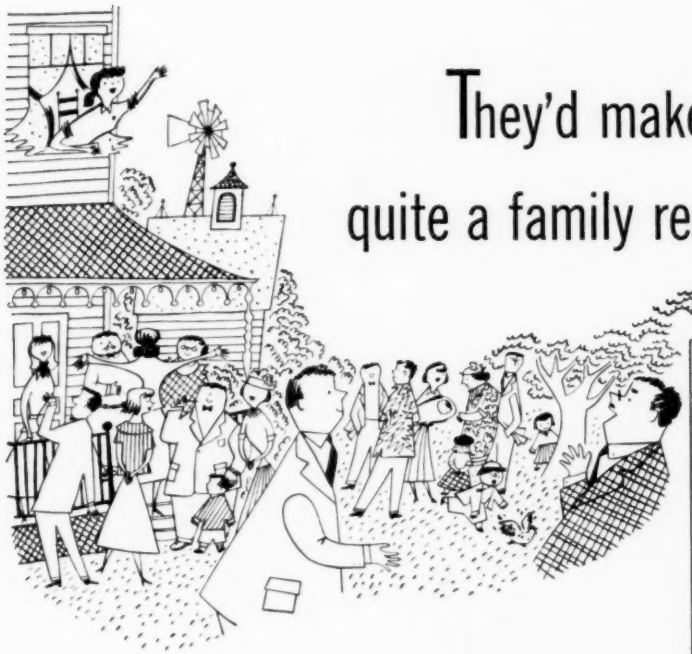

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Gastrointestinal Disorders
Cardiospasm
Pylorospasm
Spasm of biliary tract
Spasm of colon
Peptic ulcer
Colitis
Biliary dyskinesia

Allergic Disorders
Irritability
To combat stimulation of ephedrine alone, etc.

Irritability Associated With Infections

Restlessness and Irritability With Pain

Central Nervous System
Paralysis agitans
Chorea
Hysteria
Delirium tremens
Mania

Anticonvulsant
Traumatic
Tetanus
Strychnine
Eclampsia
Status epilepticus
Anesthesia

HYPNOTIC
Induction of Sleep

OBSTETRICAL
Nausea and Vomiting
Eclampsia
Amnesia

SURGICAL
Preoperative Sedation
Basal Anesthesia
Postoperative Sedation

PEDIATRIC Sedation for:
Special examinations
Blood transfusions
Administration of parenteral fluids
Electroencephalography
Minor surgery
Preoperative Sedation